

A STUDY ON VATHA PANDU

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INTRODUCTION

The Siddha Medicine is one of the oldest medical systems known to mankind. Contemporary Tamizh literature mentions the origin of the medical system from Southern India in the state Tamil Nadu, as part of the trio Indian medicines - ayurveda, siddha and unani. Reported to have surfaced more than 2500 years ago, the Siddha system of medicine is considered one of the most antiquated traditional medical systems.

"Siddhargal" or Siddhas are the great scientists of ancient days mainly from Southern India who laid foundation for this system of medication. Siddhars are spiritual adepts who possessed the ashta siddhis, or the eight supernatural powers. Sage Agathiyar is considered the guru of all Siddhars, and the Siddha system is believed to have been handed over to him by Lord Muruga, son of the Hindu God - Lord Shiva and Goddess Parvathi. So, are the siddhars the followers of Lord Shiva (Shaivaites). Agathiyar is the first Siddhar, and his disciples and other siddhars of other schools contributed thousands of texts on Siddha, including medicine, and form the propounders of the system in this world.

This disease Vathapandu correlate with Anaemia in allopathic entity. The word anaemia actually means "Lack of blood" in old Greek. This disease affects one of five people in the developed world and affects

almost everyone who suffers from malnutrition, Chronic bleeding disease. It is a common disorder in women of reproductive age and those who are pregnant as well as infants.

This disease according to Siddha concept is caused due to disturbances in blood which is one of the seven dhatus in our body. Great Siddhar Yugi muni quotes about the seven humours as follows.

சென்ம மார்ச ரீரற் தொண்ணூ ற்றுறங்கு லமாம்

செய்கின்ற வரவர்கள் கையாலேதான்

தன்னமாம் ரசமரத்தமாங்கசமு மேதை

தசை மச்சையொடு சுக்லந்தா தேழாகீ

உண்மா முருவெண்ணுஞ்ச ரீரமாகி

உயிர்சட்டை ரசஞ் சேர்ந்து ரோமமாகி

என்னவாம் வாதபித்த சேட்பமாகி

வருகின்ற சட்டத்தினட வண்மை கேளே.

The seven humours are formed from panchaboothas by pancheekaranam. Thirumoolar says this as

இரதம் உதிரம் இறைச்சி தோல் மேதை

மருவிய லத்தி வாழும் பொடு மச்சை

பரவிய சுக்கிலம் பாழாம் உபாதி

உருவம் லாலுடல் ஒன்றெனலாமே

- திருமந்திரம் 2086

This blood is subjected to various researches since ancient days. It was also believed that the man's life is decided by blood in those days. Blood acts as mirror that reflects any pathological changes in the body. According to pattinathar there are crores of uyir anukkal in blood. He says

மேவிய புன்மயிர்த் தொகையே அம்மயிர்
பாவிய தோலின் பரப்போ, தோலிடைப்
புகலிட்டுப் பொதிந்த புண்ணோ, புண்ணிடை
ஊறும் உதிரப் புனலோ, கூறு செய்து
இடையினை நிற்கும் எலும்போ, எலும்பிடை
முடைகெழு மூளை விழுதோ வழுவுழுத்து
உள்ளிடை யொழுகும் வழும்போ மெல்ல நின்று
ஊறும் புழுவின் ஒழுங்கோ நீரிடை

பட்டினத்தார்

(திருவிடை மருதூர் மும்மணிக் கோவை)

Recent researches also justifies this statement. In our body, blood represents “Appu” Kooru. Sathaga nadi says this as follows.

பாரப்பா பூதமைந்த மண் நீர் தேயு
பரிவாயு வாகாய மைந்தினாலே
சேரப்பா கடமாச்சு மண்ணின் கூறு
செறிமயிர் தோல் என் பிறைச்சி நரம்பைந்தாகும்
நேரப்பா அப்புவின் கூறு திரமச்சை
நீர்முளை சுக்கில மோடைந்தாகும்

Vatha pandu, one of the type of pandu which is caused by disturbances in blood affects many crores of people in a developing country like India. Environmental pollution, lack of a well balanced diet, infections and poisonous substances etc. are all promoters of this disease.

Though this disease can be diagnosed and treated easily if left untreated in its advanced stage it may affect important internal vital organs like heart, kidney digestive system etc.

For the above mentioned reasons I have selected to study this disease with **Thiratcha Choornam. (Agasthiar Pari Pooranam 400).**

ANEMIA

Introduction

Anemia is defined as a hemoglobin concentration in blood below the lower limit of the normal range for the age and sex of the individual. In adults, the lower extreme of the normal hemoglobin is taken as 13.0 g / dl for males and 11.5 g / dl for females. Newborn infants, have higher hemoglobin level and, therefore, 15g / dl is taken as the lower limit at birth, whereas at 3 months the lower level is 9.5 g / dl. Although hemoglobin value is employed as the major parameter for determining whether or not anemia is present, the red cell counts, hematocrit (PCV) and absolute values (MCV, MCH and MCHC) provide alternate means of assessing anemia.

Generally, anemia occurs because of :

1. Decreased production of RBC,
2. Increased destruction of RBC or
3. Excess loss of blood from the body.

All these incidents are caused either by inherited disorders or environmental influences such as nutritional problem, infection and exposure to drugs or toxins.

Prevalence of Anemia

Anemia is one of the most common health problems in India. The problem is much more in rural than the urban areas. The high-risk groups for anemia are pregnant and lactating females and children. Prevalence in this subgroup has been found to vary from 50-90% in different parts of India. Almost all interventions at national and local level have focussed predominantly on these groups. Reliable data on the prevalence of anemia in adult population (non-pregnant females and adult males) is not available. During an epidemiological study on prevalence of hypertension in rural population,⁴ we studied prevalence of anemia among adult individuals.

AIM AND OBJECTIVES

The aim of the work is to evaluate the clinical efficacy of the trial drug (Thiratcha Choornam) in vatha pandu, one of the types of Pandu.

The Vatha Pandu to be correlated with clinical condition called “Anaemia”.

I has choosen Vatha Pandu for the dissertaton study because It affects all age from children to old age.

The disease is diagnosed in (1) Clinical methods of Siddha Medicine and (2) Relevant modern diagnostic methods.

The selection of cases is from the out-patient department and in-patient department of Government Siddha Medical College.

This dynamic approach deals very much in detail, with the work done with devotion and dedication to bring out following objectives.

1. To collect through study of various literatures dealing with definition, classification, sign and symptoms, naadi, diagnosis, diets.
2. To expose the reliability of siddhar’s diagnostic principles.
3. To know the clinical efficacy of the trial drug.
4. Bio chemical investigations and pharmacological action of the medicine tried on this disease.

5. To have an idea of the incidence of the disease with Sex, age, habitat, occupation, income, social status, diet etc.

ABSTRACT

Since being the commonest disease in the society, number of suffers increasing day by day, I has chosen the disease “VATHA PANDU” for his dissertation work. The evidence of the disease VATHA PANDU is derived from “Yugi Vaidhya Chinthamani – 800”. The signs and symptoms mentioned in Yugi Vaidhya Chinthamani book closely resembles with that of Anemia in modern medicine.

20 Inpatients and 20 Out patients of both sexes were selected. They were administered with the trial medicine “Thiratcha Choornam” 2 gms BD with Ghee during the whole study period. Agathiyar Paripooranam (page no 57, 58) (Rathina Nayagar & Sons, Chennai – 79, 1995).

The trial medicine was subjected to biochemical and pharmacological analysis.

At the end of the trial study, the majority of the cases showed good results.

REVIEW OF SIDDHA LITERATURE

SIDDHA ASPECTS

சிவனென்னச் சீவனென வேறில்லை

- திருமூலர்

அவனே தானாகிய அந்நெறி

ஏகனாகி இறைபணி நிற்க

மலமாயை தன்னோடு வல்லினையின்றே

- சிவஞான போதம்

Siddha system is said to have originated from LORD SIVA AGASTHIYAR is the first disciple of Siva, nobody deny the fact that Agasthiyar was Guru and pioneer to propagate this siddha system of medicine.

The siddha system of medicine is based on the Tridhosha theory according to which the human system is medicated by three vitiating elements (or) Thatus. The functional units of living beings namely vatham pitham and kabam. Diseases are produced by disequilibrium of these three vitiating elements, which may be due to various causes like dietetic regimen, life style patterns and the ecological imbalance etc.

The disease pandu has its historical importance. The word pandu has been derived from HINDU EPIC “MAHABHARATHAM” where the father of five heroes “Pancha Pandavar” is pandu. It is said that this man, when born was very pale and anaemic and hence this condition was named after him as pandu.

Synonyms of Pandu

Venmai noi, Velluppu noi, Ven pandam.

Iyal (Definition)

Pandu is the disease of Raktha thathu characterized by pallor of skin mail beds and conjunctivae.

The great siddhar Agathiar define pandu in the following verses.

கழிவாகுந்த தேகமப்பா காணத்ததுவத்தாய்

வற்றிவிடு மன்னவாசல் கேட்கில்

பழிகாரர் முகத்தினில் முழியார் போலே பாண்டமெலாம்

வெளுக்கடித்தது ரத்தம்

- அகஸ்தியர் வைத்ய காவியம்

தேகத்தில் இரத்தம் வற்றித்

தீங்கான விந்த நோய் காணுமப்பா

- அகஸ்தியர் குணவாகடம்

Formation of Raktham according to Siddhars

As the digestion takes place in the body “Rasa thathu” is formed on the very first day on the second day “Raktham” is formed from the Rasa thathu. And from Raktham masmisam is formed, from this kozhuppu is formed, from kozhuppu enbu is formed, from enbu moolai is formed, from moolai sukilam is formed on the 3rd, 4th, 5th, 6th and 7th day respectively.

It is to be noted that the nutrients absorbed after digestion are responsible for the metabolic functions and the formation of blood.

Noi Varum Vazhi (Aetiology)

a) According to YUGIMUNI, AETIOLOGY is defined as follows.

அறிந்துமே உற்பத்தி சொல்லக் வேளாய்

அதிசார மலமிளகி யெந்நே ரந்தான்

பிறிந்துமே புளியுப்பு பெருத்தாலும்

பெத்தமா மக்கனியி லிருந்தலாலும்

மிறிந்துமே தாம்பூல மிக அருந்தலாலும்

மீறியே மதுக்களைத் தான் புசித்தாலும்

பறிந்துமே பகல் நித்திரையே செய்தலாலும்

பாண்டு வந்து பாரிலுள்ளோர் படும் பாடாமே.

பாடான பஞ்சுதனை திருடினோர்க்கும்

பாங்கான சிவ துயிலைதத் திருடினோர்க்கும்

மாடான பசுவதைப் பட்டினியாக

வைக்கின்றோர் மறை வழியில் நடந்திடாதார்

காடான வாரணியந் தனிற்ப றித்துக்

கடுவதைகள் செய்கின் றோர்கள் காணாத

கேடான பழிசொல்லிக் குடி கெடுக்கும்

கொடும் பாவி பாண்டு வினாற் குதி கொள்வாரே.

- யுகி வைத்திய சிந்தாமணி.

Yugi says that the faulty way of living brings about this pandu condition while listing out the wrong life styles, he says about excessive in take of food stuffs with salt and sour items, staying in hot conditions excessive in take of pan and nap etc.

b) Cause of Pandu from Thanvanthiri Vaidyam

திருந்திடும் பாண்டு ரோகங் சேர்ந்திடுங் குணத்தைக் கேளாய்

இருந்திடும் வாத பித்தச் சிலேற்பன மிவைதான் மாறும்

பரிந்துதா னொன்றோ டொன்று பொருந்துவதாலு மண்ணோ

டருந்துவதாலும் பாண்டு அணைந்திடு மென்னலாமே

ஆகிய மூலந்தன்னி லணைந்தவுட் டணத்தினாலுற்

தேகபோஷ ணையுள்ளார்க்குத் தரித்திரஞ் சேர்தலாலும்

வேகமாந் திரிதோஷங்கள் மிஞ்சியே பாண்டு வாமே

- தன்வந்திரி வைத்தியம்

Imbalance between the three thathus perversion of appetite in the form of geophagia excessive heat accumulation due to altered abana, sorrow, psycho social factors and also closely accompanied with several transmitted disease.

Pandu – Kanma Noi

- c) IN AGASTHIAR PARI POORNAM – 400 pandu is considered to be the one among the kanma noi.

செல்லாத கடிவிஷங்கள் குன்மம் பாண்டு

துயர்தீராக் கன்மவினை செய்த பாவம்

எல்லோரும் நகைக்க உடும்பெடுத்த பாவி

இன்னமுண்டு விபரமாய் உலகிற் கேளே.

The same thought was again confirmed in Agasthiar Vaidyam Mun – 80.

குயல்வாய் குஷ்டம் சயங்குன்ம நீரிழிவு சுரக்கிராணி

நீரடைப்பு பாண்டு மூலவாய்வு

கன்மவாயு வருங்கண்ணில் குந்தாய் கடினந் தசவாங்வு

காணவாக முன்செய்த உயிர்களும் வினைதானே.

d) AGASTHIAR GUNAVAGADAM says

கொள்ளடா அபக்குவ போசனத் தினாலும்

குடி கெடுத்த பெரும்பாடு கிராணியாலும்

கள்ளடா கருப்பத்தின் கிராணியாலும்

கனமான ரத்தத்தின் போக்கினாலும்

அள்ளடா அதியாம கவனத் தாலும்

அளவற்றவி சாரந்தானடையும் போதும்

தெள்ளவே தேகத்தில் ரத்தம் கெட்டி

தெளிவான பாண்டதுவு முண்டாம் பாரே

- அகஸ்தியர் குணவாகடம்

Agasthiyar says “wrong food habits and injudicious diet, menorrhagia in females, chronic diarrhea, blood loss due to various aetiology, stress and strain are some of the causes of pandu.

e) Due to worm infestations

வயல்தனிலே பூநாக மண்ணைத் தானே

வருந்தியது புத்துபோல வத்தையாகும்

பயில் மொழியீர் தேகத்தில் கிருமிதானே

- குருநாடி

The above stanza from Gurunadi illustrate the worm infestation.

Pathological loss of blood may occur due to various factors. One among them is worm infestation lead to chronic blood loss from the intestinal region. Hence heavy infestations are bound to cause anaemia.

f) Another concept from THERAYAR VAGADAM denotes the causative factor of pandu noi.

கருதிய மீனின் முள்ளும், கலந்துமி நெய்யின்
மருவிய எலும்புங் கல்லும் மங்கையர் மருந்தீடும்
பருகிய பழஞ்சோற்றாலும் பழமல நிறைகையாலும்
மருகிய மயிர்களாலும் வந்திடும் நோயிதாமே
புறவரை யுண்கையாலும் போக நீருண்கையாலும்
சுருளவே முடக்கிக் கொண்டு முறக் கிடக்கை யாலும்
மருளவே மேடு தன்னில் மனமுறக் கிடக்கையாலும்
பெருக வெண்டொடியினாலும் பிறக்க நோயென்று காணே

Therayar Vagadam says, thorn of fish, paddy ban, bone, stones, old rice, hairs in food are the dietetic causes.

Secondly severe constipation, drinking of polluted water sleeping in a abnormal posture then over indulgence with ladies are all bring out veluppu noi.

- g) In AGATHIAR VAGADAM the causes of pandu noi is mentioned as follows.

நாமொன்று சொல்லுகிறோம் பாண்டு வந்த
நலமான கருமமது சொல்லக் கேளு
தாமென்ற தாய் தந்தை மனம் நோகச் செய்தல்
தரணிதனி லுள்ளவர்க்கும் அன்பே சொல்ல
லாமென்ற விருப்பழித்தல் செவி சுவாச
மற்றும் பொய் சொல்லாங் காரஞ் செய்தல்
வேமென்ற கருமமது பித்தமேறி
வெறுத்ததடா வெப்பு மிஞ்சிக் கை காலோய்வே

By which the irresponsible and disobedience parents, unhospitality to people, speaking lie, gets anger on others stimulates pithadhosha which results in pandu noi.

- h) In THERAYAR MAHA KARISAL Aetiology of pandu is defined as follows

வாத பாண்டு நோயே தானும் வந்திடும் பரிசு கேளாய்
சாதம் நீரிச்சையற்றுத் தன்னிறங் கருதி வாடிச்
குதுசேர் சொருபமுண்டாய்ச் சுரோணிதம் வற்ற வற்ற
ஆதாரவாமனைத்தும் அதிசய மாகுங் கண்டாய்

- தேரையர் மகா கரிசல்

NOI ENN (CLASSIFICATION)

YUGI discuss this disease under five pandu noi

சுறவே பாண்டுவிடப் பெயரைக் கேளாய்
குறிப்பாக வைந்து விதமாகும் பாரு
வாரவே வாதமாம் பாண்டி னோடு
மார்க்கமாம் பித்தத்தின் பாண்டு தானும்
தேறவே சிலேட்டுமமாம் பாண்டு தானும்
திரிதோடப் பாண்டோடு விட பாண்டாகும்.

THANVANTHIRI classified into five types of pandu noi

வியத்திரி தோஷப் பாண்டு வெளும் பித்த சிலேத்மப் பாண்டு
பயித்திய வாதபாண்டு பகர் சந்நி வாத பாண்டு
நயப்புறும் பாண்டு வேழின் குணத்தை நான்நவிலலுற்றேன்.

AGASTHIYAR classified into five types of pandu noi

பாரடா பாண்டு வகை சொல்லக் கேளாய்
பிரிவான பாண்டதுதா னஞ்சே யாகும்
வாரடா வாத பித்தம் சீத பாண்டு
வகையான விடபாண்டு மிருந்திகா பாண்டு

Our Siddhars classified this pandu noi under three dhoshos and have given names according to their concept. The signs and symptoms of classified disorders are almost identical in the description of the disease.

Classification of pandu noi

Yugi Chinthamani

Vatha pandu

Pitha pandu

Kapha pandu

Mukkuutra pandu

Visha pandu

Thanvanthiri Vaithiyam

Vatha pandu

Pitha pandu

Kapha pandu

Mukkuutra pandu

Pitha Vatha pandu

Pitha kappa pandu

Sanni patha pandu

Vaidyasara Sankirakam

Vatha pandu

Pitha pandu

Moola pandu

Moola pitha pandu

Visha pandu

Agasthiyar Gunavagadam

Vatha pandu

Pitha pandu

Moola pandu

Nanju pandu

Miruthiya pandu

Jeeva Rakshamirtham

Vatha pandu

Pitha pandu

Kapha pandu

Mukkuutra pandu

Nanju pandu

Mirthikapuktha pandu

Alimuga pandu

T.V.Samba Sivam

Pillai Dictionary

Vatha pandu

Pitha pandu

Kapha pandu

Mukkuutra pandu

Oothu pandu

Neer pandu

Eri pandu

Visha pandu

Roga Nirnaya

Saram

Vatha pandu

Pitha pandu

Kapha pandu

Mukkuutra pandu

Visha pandu

Anuba Vaidya

Deva Ragasiyam

Vatha pandu

Pitha pandu

Kapha pandu

Mukkuutra pandu

Nanju (or) visha

Pandu

Mirthikapuktha

Pandu

Various Siddha literature enumerate the types of pandu in different classification but all of these consists Vatha pandu as one of the variety.

Noi Kurikunangal (Clinical features)

a) Premonitory Symptoms

Pandu patients exhibit the following symptoms from their initial stage of development itself. The patient experience insidious onset of fatigability lassitude, difficult in breathing on exertion, diminished vision, faintness, palpitation, palloriness of the skin.

b) Signs and Symptoms of pandu

According to Gunavagadam

Palloriness of the face, eyes, lips, tongue, pallor of the skin, pallor of the nail, oedema of the feet due to CCF.

உண்டாகும் வேளை தன்னில் தேக நேர்மை

உறுதியாய்ச் சொல்லுகிறேன் நன்றாய் பாரு

குண்டான முகம் கண்கள் உதடு நாக்கு

குறிப்பான வாய்வேகும் தேக முற்றும்

வெண்டாக வேயுலர்ந்து வெண்மை யாகி

விரல் நகங்கள் முழுவதிலும் ரத்தம் வற்றி

கண்டான கால்கள் தான் தணிந்து நிற்கும்

கருவான நாடியது மெதுவாய்ப் போமே. (23)

போமேதான் தீபனங்கள் மட்டுப்பட்டு

பொலிவான கண்விழிகள் பெருத்துத் தோன்றும்

ஆமேதான் அசதியு மாயாசங் கண்டு

அவர் நடையும் தளர்ந்து பெருமூச்சுக் கண்டு

மூவேதான் மூர்ச்சையுடன் மார் துடித்து

முடிவான கணுக்காலில் விக்க முண்டாய்

தாமேதா னிருதயத்தின் வதனந் தன்னிற்

தரத்தி நிகர் சத்தமது கேட்கும் பாரே.

அகஸ்தியர் குணவாகடம்

The features of Vatha pandu are parallel with that of Yugimuni's features.

Vatha pandu as told in Yugimuni's Vaidya Chinthamani's

In Association with general signs and symptoms of pandu due to vatha vitiation the below mentioned signs and symptoms are also present. They are abdominal discomfort, loss of appetite, increased thirst, shivering of the body with generalised oedema.

கொள்ளவே வாதபாண்டு ரோகங் கேளாய்

குடல் பிரட்டி யடி வயிறு தான் வலிக்கும்

தன்னவே தாகமொடு பசியு மில்லை

தழலான சுரசுரப் பாகத் தானும்

நள்ளவே நரம்பெல்லாம் கறுப்பு மாரும்

Roga Nirnaya Saram

Pain all over the body, tremor, discolouration of nail, conjunctiva.

Mukutra Verupadugal (Pathology of vatha pandu)

Our Siddha medicine is based upon thridoshic theory. Due to intrinsic and extrinsic factor, the pitha in the body gets altered so digestion affected by the affected pitha thathu. In the blood stream the ranjagam which is the branch of pitha which gives colour to the blood is unable to entertain these ill digested and improperly assimilated nutrients with Raktha thathu. So a defect manifest in the haemopoiesis. So vatha pandu produces pallor of the tongue, nailbeds, conjunctiva, and skin etc.

The deterioration of thathu with pitha and vatha, produces oedema. These altered tridosha finally altered the normal structure and functions of the seven thathus.

Agasthiar in his vadha kaviyam say that for upsetting and deranging all the functions of vatham and pitham is mainly due to the dietetic factors.

PINIYARI MURAIMAI (Diagnosis)

Piniyari muraimai is the method of diagnosing the disease affecting the man. It is based upon three main principles.

They are

1. Poriyalarithal
2. Pulanalarithal
3. Vinathal

Pori is considered as the five senses of perception namely Nose, Tongue, Eye, Skin and Ear. While pulan are five objects of senses. They are Smell, Taste, Sight, Sensation and Sound. Vinathal is obtaining the information regarding the history of the disease its clinical features etc from the patient or his immediate relatives who are taking care of him, when the patient is not in a position to speak or if the patient is a child.

The above principles correspond to the methodology of inspection, palpation and interrogation of modern medicine

Alavai

Alavai is a parameter through which one can assess the real properties merits and demerits of the things using the five senses as the instruments.

Alavai is very much useful in diagnosing vatha pandu as follow.

1. Kandal (Observation)

By observing the palloriness of skin, conjunctiva, nail beds, mucous membrane of lip and tongue.

2. Karuthal (Inference)

Patient complaints of fatigue, palpitation, dyspnoea on exertion the physician can have a clue for the diagnosis of vatha pandu.

3. Urai (Authority)

In addition to the above manifestation the kabha, kabha vatham, kabha pitham, vatha kabham felt in the vatha pandu patient further confirms the diagnosis.

This has been quoted in

வாதத்தில் சேத்துமமாகில் வலியோடு வீக்கமுண்டாம்

பேதித்துத் தலையிடித்துப் பிணங்கிய குணங்கள் வேறாய்

தீதற்று மெய்வெளுத்துத் திடமுடனசனஞ் செல்லா

பேதித்து நாவு பேசும் பெருகவே வீக்கமுண்டாம்

- அகஸ்தியர் நாடி.

உண்டாயோ சேத்துமத்தில் வாதநாடி
கலந்திடுமேல் பாண்டு பிறக்குந்தானே
கண்டாயோ சிலேற்பனத்தில் வாதநாடி
கலந்திடுகில் வயிறு பொருமல் கனத்த வீக்கம்
உண்டாலோ ஓங்காராஞ் சக்தி விக்கல்
உறுதிரட்சை வாய்வு வலி சந்தி தோடம்
விண்டாலோ யிளைப்பிருமல் சோபை பாண்டு

- சதக நாடி நூல்

தானமுள்ள சேத்து மந்தானிளகில் வெப்பு,
சயமீளையிருமல் மந்தார காசம்
ஈனமுறுஞ்சந்திவிடதோடம் விக்கல்
யிருந்ரோகங்கரப்பான் விரண தோடம்
மானனையீர் குலை திரள் வியாதி வீக்கம்
வருஞ்சத்தி சுவாசம் நெஞ்சடைப்பு, தூக்கம்
ஏனமுறுங் காமாலை பாண்டு சோபை.

- சகடி நாடி நூல்.

இடமான சேத்துமத்தில் பித்தநாடி
எழுந்தனுகில் விடமுடனே வீக்கமுண்டாம்
திடமான குளிர் காய்ச்சல் மஞ்சள் நோவுந்
தேகத்திலுளைச்சலிளைப் பிருமல் வாந்தி
விடமான நெஞ்சடைப்பு சுவாசம் விக்கல்
வெகு கரமும் நாவறட்சி பாண்டு ரோகம்

- சதக நாடி நூல்.

Enn Vagai Thervugal

Diagnosis is confirmed by Enn vagai thervugal, which are named as follows.

Naa,

Niram,

Mozhi,

Vizhi,

Malam,

Moothiram,

Naadi

Sparism

This is evident from the version of Theraan one of the great Siddhars.

நாடிப் பரிசம் நா நிறம் மொழி விழி

மலம் மூத்திரமிவை மருத்துவராயுதம்

மெய்க்குறி நிறம் தொனி விழி நா இருமலம்

கைக்குறி.

- தேரையர்

In Agasthiyar vaidhya vallathi – 600 Ennvagai thervugal has been mentioned as Attavitha paritchai

தொக்கலுற்று அட்டவிதப் பரீட்சை தன்னை

துலக்கமுறும் பண்டிதரே தெளிவதாகப்

பகுக்கரிய நாடியைநீ பிடித்துப் பாரு

பகர்கின்ற வார்த்தையைப் பார் நாவைப்பாரு

வகுக்கரிய தேகமெனத் தொட்டுப் பாரு

வளமான சரீரத்தின் நிறத்தைப் பாரு

சகிக்கரிய மலத்தைப்பார் சலத்தைப் பாரு

சார்ந்த விழிதனைப் பார்த்து தெளிவாய் காணே.

Agasthiyar Vaidhya

Vallathi – 600

நாடியால் முன்னோர் சொன்ன நற்குறி குணங்களாலும்

தீடிய விழியினாலும் நின்ற நாக் குறிப்பினாலும்

வாடிய மேனியாலும் மலமோடு நீரினாலும்

சூடிய வியாதி தன்னை சுகம் பெற வறிந்து சொல்லே.

- அகத்தியர் நாடி.

Naa (Tongue)

The colour change according to vatha pitha and kabha, mukutra disease, dryness or wet, coated or not, excessive salivation, redness, ulceration, pallor, yellowish discolouration of the tissues, neoplastic growth, conditions of teeth its colours, condition of the gums, taste, loss of appreciation, speech, deviation of the mouth angle, movements of the tongue can be made out.

In vatha pandu noi, the tongue was pallor moist, and glossy and the sense of taste would be altered.

Niram (Colour of the skin)

Colour indicating vatha, pitha, kabha and three dhoshas, yellow or pallor or redness of the skin, any bluish discolouration.

In vatha pandu noi, the colour of the skin, tongue, nailbeds and conjunctiva were very pale.

Mozhi (voice)

Clarity of speech or any disturbance, loud voice, slurring, crying, talk induced by hallucination, breathlessness can be made out.

In vatha pandu noi patients were spoken in diminished voice. Some were in breathlessness.

Vizhi (Eyes)

Any abnormal colour change indicating the three doshas derangements, pallor, excessive lacrimation, sub conjunctival bleeding, drooping of eye lids, visual disturbance, any specific diseases of the eyes.

In all vatha pandu noi patients pallor of the conjunctiva was observed.

Malam (Faeces)

Quantity, semisolid, colour, odour, froth, abnormal consistency indicating indigestion, frequency, constipation, sulphur smell, foul smell etc. are observed.

In vatha pandu noi, constipation is usually encountered.

Moothiram (Urine)

Quantity, colour, Odur, froth, frequency, deposits, presence of abnormal constituents such as sugar, protein etc.

Examination contains two phases Neerkuri (The colour indication
Neikuri (The oil indication)

Neerkuri

அருந்துமாறிரமும் அவிரோதமதாய்
அ.கல் அலர்தல் அகாலவூன் தவிர்ந்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாலியே காது பெய்
தொருமுகூர்த்தக் கலைக்குட்படு நீரின்
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே

The patient must take well cooked food on the previous day. The intake must be proportionate to the degree of his appetite. Food should be taken at appropriate time. He must have sound sleep on that night. The urine is collected on the dawn of the next day in a glass container and closed immediately to prevent evaporation. The specimen must be examined within 1½ hours. This procedure should be followed strictly in order to get accurate reading of Neerkuri and Neikuri.

In vatha pandu not straw yellow coloured urine was observed.

Neikuri

நிறக்குறிக் குரைத்த நிருமாண நீரிற்
சிறக்க வெண்ணெய்யோர் சிறுதுளி நடுவித்
தென்றுறந் திறந்தொலி யேகாதமைத்ததி
னின்றதிவலை போம் நெறிவிழியறிவும்
சென்றது புகலுஞ் செய்தியை யுணரே

The specimen is kept open in a glass dish being exposed well to sunlight. But it should not be disturbed by the movement of the wind. Then add one drop of gingely oil by a glass rod. Observe keenly the position and spreading of the oil drop dropped on the surface of the urine.

In vatha pandu not snake like spreading was observed.

1) Sparism (palpation)

The following points are elicited by sparism, temperature of the skin any abnormal growth, hyper sensitiveness dryness of skin ulcers, oedema, etc.

In vatha pandu noi the sparism was warm.

Naadi

Naadi is the vitiating element of the body which are vatham, pitham and kabam. Naadi is the vital force. The examination of naadi has been recognised as one of the principal means of diagnosis and prognosis of the disease from time immemorial. The great Siddhar Thiruvalluvar says,

நோய் நாடி நோய் முதல் நாடி அது தணிக்கும்

வாய் நாடி வாய்ப்பச் செயல்

Any change in the three doshas is best diagnosed by feeling the Naadi.

மிகினும் குறையினும் நோய் செய்யும் நூலோர்

வளி முதலா எண்ணிய மூன்று

மூன்றி லொன்றுயர்ந்ததை முன்னரறிந்து

முந்தியதனை யொழித்திடு மருந்திடு

தணியும் நோயின் தந்திர மிதுவே

பேணிக் கணித்திடின பிறவாய்ப் பிங்குணம்.

At the lower end of radius, Naadi is felt as vatham pitham and kabam with the tips of index, middle and ring fingers respectively.

They are formed by the combination of

Edakali	+	Abanan	-	vatham
Pinkalai	+	Piranan	-	pitham
Suzhumunai	+	Samanan	-	kabam

The ratio between vatham, pitham, kabam, is 1:½:¼ respectively.

வழங்கிய வாதம்மாத்திரை யொன்றாகில்

தழங்கிய பித்தந்தன்னலரை வாசி

அழங்குங்கபந்தானடங்கியே காலோடில்

பிறங்கிய சிவர்க்குப்பிச கொன்றுமில்லையே

(குணவாகடம்)

மெய்யளவு வாதமொன்று

மேல்பித்தமோரரையாம்

ஐயங்காலென்றே அறி.

(கண்ணுசாமியம்)

On the basis of the examination of the senses and on the basis of eight special examination and interrogation all the details of the disease factor are collected and their final diagnosis is confirmed with those finding made on naadi paritchai.

In vatha pandu noi the following types of naadi are seen commonly.

1. Kabam
2. Kaba vatham
3. Kaba pitham
4. Vatha kabam.

Besides Ennvagai thervugal, a disease can also be diagnosed by means of other methods namely Thinaigal, Paruvakalanga, Uyirathukkal, udal thathukkal. Hence a through knowledge about the disease can be attained systematically and properly in siddha system of medicine. A combination of all these diagnostic criteria are very helpful to attain a proper

diagnosis with a full complete entity based on basic principles of Siddha science.

Thinaigal

Nilam is classified into five types, depending on the surrounding vegetation, landscape and ecological stage, study of five places is very much necessary as some diseases are common in particular land.

1. Kuringi : Mountain and its surroundings liver diseases and flurosis are common
2. Mullai : Forest and its surroundings.
3. Marutham : Field and its surroundings. The ideal place for healthy living.
4. Neithal : Sea and seashore liver disease occur in combination with other diseases.
5. Palai : Desert and its surroundings vatha, pitha and kaba diseases occur.

In all five types of lands “Vatha Pandu” is probable of occurrence

Paruvakalangal

Siddhars have classified a year into six seasons each consisting two months. They are

1. Kaar kalam : Avani and Purattasi
2. Koothir kalam : Iyppasi and Karthigai
3. Mun pani kalam : Margazhi and Thai
4. Pinpani kalam : Maasi and Panguni
5. Elavenir kalam : Chithirai and Vaikasi
6. Muthuvenir kalam : Aani and Aadi.

Some of the diseases are commonly prevalent during a particular paruvakalam and a study of it will be of much use for diagnosis.

In all six seasons “vatha pandu” is probable of occurrence.

Udal Vanmai (Immunity)

Siddhars classified this udal vanmai into three types. They are

1. Iyarkai Vanmai
2. Seyarkai Vanmai
3. Kala Vanmai

Iyarkai Vanmai

Natural immunity of the body caused by mukkutram right from birth onwards.

Seyarkai Vanmai

Improving the health by intake of nutritious food materials, activities and medicines.

Kala Vanmai

Development of immunity according to age and environment.

When udal vanmai is affected, there may be possibility of “vatha pandu”.

Uyir thathukkal

Three initiating elements are the functional units of the body, namely vatham, pitham and kabam.

Vatham

Vatham is the kinetic energy, which influences all motions.

Vatham is located in the abanan, motion, edakalai, spermatic cord, pelvic bone skin, nerves, joints, hair follicles and muscles, bones air and thigh. It is classified into ten types. The are

1. Piranan (Uyirkkal)

This control knowledge, mind and five objects of senses useful for breathing and digestion.

2. Abanan (Keezhnokkukal)

It expels stools, urine, semen and foetus.

3. Udhanan (Melnokkukal)

It responsible for all upward visceral movements such as vomiting, eructation, nausea.

4. Viyanan (Paravukal)

It is used to feel all type of sensations. It carries nutrients to all over the body. Flexes and extends the movable joints.

5. Samanan (Nadukkal)

It prevents and exhibits the over activity of other vayus.

6. Nagan

It controls the blinking of eyes.

7. Koorman

Responsible for vision, yawning and lacrimation.

8. Kirukaran

Responsible for salivation, nasal secretion and appetite.

9. Thevathathan

Sleep, fatigue, tiredness, anger are caused by Thevathathan.

10. Thananjeyan

It produces oedema of the body and hyperacusis in the ears. It escapes on the 3rd day after death by bursting the cranium.

In case of “VATHA PANDU”

- | | | |
|-----------------|---|--|
| 1. Abanan | : | (expells stools) |
| 2. Viyanan | : | (It carries nutrients all over the body) |
| 3. Udhanam | : | (Vomiting) |
| 4. Samanan | : | (Due to other vayus are affected) |
| 5. Kirukaran | : | (Appetite affected) |
| 6. Thevathathan | : | (Tiredness and sleep affected) |

Above the vauyus are mainly affected other vauyus are not affected commonly.

Pitham

Located in urinary bladder, heart, head, pingalai, umbilicus, abdomen, piranan, stomach, blood, sweat, skin and eye.

Pitham is classified into five types. They are

1. Anal Pitham : This gives appetite and helps for digestion.
2. Ranjaga Pitham : It gives colour to the blood.
3. Pirasaga Pitham : It gives complexion to the skin.
4. Alosaga Pitham : It is responsible for vision.
5. Sathaga Pitham : It is responsible for the action what we think.

In the case of VATHA PANDU

1. Anal Pitham : (Appetite and digestion)
2. Ranjagam : (Colour to the blood)
3. Pirasagam : (Complexion of the skin)
4. Sathaga Pitham : (Unable to carryout regular works properly)

These are mainly affected.

Kabam

Located in samanana, semen, head, tongue, fat, bone marrow, blood, nose, chest, nerves, bones, brain, large intestine, eye, stomach and pancreas.

Kabam is classified into five types. They are

1. Avalambagam

Heart is the center for avalambagam. It controls all the other kabams.

2. Kilethagam

Stomach is the center for kilethagam. It gives moisture and helps in the digestion.

3. Pothagam

Tongue is the center for pothagam. It is responsible for the sense of taste.

4. Tharpagam

Head is the center for Tharpagam. It gives cooling to the eyes.

5. Santhigam

It lies in the joints and is responsible for the action of joints. In the case of “vatha pandu”

Kilethagam : (Digestion)

Pothagam : (Taste sensation)

Avalambagam : (It controls all other kabam)

These are commonly affected.

Ezhu udal kattugal

These are seven basic principles, which constitute the entire body.

1. Saram

Strengthens the body and mind.

2. Senneer

It is responsible for knowledge, strength, boldness and healthy complexion.

3. Oon

It gives structure and shape of the body and is responsible for the movements of the body.

4. Kozhuppu

It lubricates the joints and facilitates their functions.

5. Enbu

It protects all the internal organs and gives the structure to the body.

6. Moolai

Present inside the core of the bones and gives strength.

7. Sukkilam / Suronitham

Meant for reproduction (male and female respectively)

In the case of “vatha pandu”

- | | | |
|-------------|---|---------------------|
| 1. Saram | - | Tiredness. |
| 2. Senneer | - | Reduced haemoglobin |
| 3. Oon | - | Movements affected |
| 4. Kozhuppu | - | Functions affected. |

These are mainly affected other udal kattugal are not affected commonly.

Noi Nithanam

Prognosis of vatha pandu noi.

Yugimuni says vatha pandu noi is curable.

The prognosis of pandu becomes grave if diarrhea occurs. This is stated with the prognosis of other disease as follows.

பாண்டு பிரமேகம் பன்வாத சூலை குன்மம்

வேண்டா சயஞ் சன்னி வெண்சோபை – நீண்ட

அதிநீரே காமாலை யான பிணி தம்மு

ளதி சாரமா காதறி

The other condition stated are piramegam, vatha soolai, gunmam, kshayam, Janni, Sobai, mathumegam and kamalai. This is stated in sathaga nadi as

தானான பிரமேகம் வாதசூல

சார்வான நீரிழிவு குன்ம ரோகம்

மானான சயரோகஞ் சன்னி தோடம்

யடுத்த விடங்காமாலை பாண்டு சோகை

மானேகேள் கபரோக மந்திர வியாதி

மஞ்சள் நோய் குலை நோவு பயித்திய ரோகம்

ஊனாகும் வருமிடத்தில் யதிசாரங்கள்

உண்டாகில் யசாத்தியமா முறுதிதானே

In pandu noi and in certain other disease if a combination of emaciation, dyspnoea and hiccough occur, the patient will die. In sathaga naadi it is stated as follows.

தொகுத்திட்ட நீரிழிவு மேக சூலை

சுரலிக்கஞ் சந்நிவலி தோடமாந்தம்

மிகுத்திட்ட கிராணியதிசாரம் வாதம்

விடபாகற் திரள் பாண்டு சோகை காமாலை

வகுத்திட்ட பெரும்பாடு மஞ்சள் நோவு

வாய்வு ரத்தபித்தமுடன் பல நோவுக்கும்

பகுத்திட்ட யிளைப்புடனே சுவாசம் விக்கல்

பற்றினால் மரணமென்று பகுத்துச் சொல்லே

Kannusamiyam quoted that scanty micturition in pandu noi will cause the death,

வெப்புப் பிணியதனில் வெம்மேகத்தால் வருந்தின்

தப்பு மிகை நீரே தானிறங்கின் - செப்பும்

கிராணியில் பாண்டில் கிளர்நீர் சுருங்கில்

பிராணன் பிரியுமெனப் பேசு

Maruthuvam

வைத்தியச் செயல் வைத்தியமாமே

- திருமூலர் 8000

So our Siddha treatment is not only for complete healing but also for the prevention and rejuvenation. This is said as follows:

Kappu (Prevention)

Neekkam (Treatment)

Niraivu (Restoration)

Siddha system has unequivocally stated that even during the time of conception. Some edfects creep into the fertilized embryo. The defects form the basis for the manifestation of certain constitutional diseases later on during the existence of the individual.

Diseases are produced by the unequilibrium of three thathus which may be due to various causes like diet, life style patterns, mental and physical activities.

மிகினும் குறையினும் நோய் செய்யும் நூலோர்

வளி முதலா வெண்ணிய மூன்று

- திருக்குறள்

When treating for cure of the diseases the following principles must be noted.

நோய் நாடி நோய்முதல் நாடியது தணிக்கும்

வாய்நாடி வாய்ப்பச் செயல்

உற்றானளவும் பிணயறவுங் காலமும்

கற்றான் கருதிச் செயல்

- திருக்குறள்

So it is essential to know the disease and the cause for the onset of disease the nature of the patient, the severity of the illness, the seasons and the time of occurrence of the disease must be observed.

LINE OF TREATMENT FOR VATHA PANDU

The aim is to normalize the vitiated mukkutram, vayus and the affected Raktha thathu. As this disease is caused by the deterioration in Ranjaga pitham, effective medicinal preparations have to be administered in the beginning itself to raise the Raktha thathus achieve normal function of it.

Before starting the actual treatment, the presence of absence of toxins in the body produced due to derangement of three thathus. This explained in siddha as follows.

வாந்தியால் பித்தற் தாமும்

பேதியால் வாதற் தாமும் நசிய

அஞ்சனத்தால் கபந்தாமும்

Usually for pitha disease emetics are to be given to alter the deranged pitham. But there are some exceptions to this rule for instance in veluppu not since the patient is already weak and drowsy. The administration of vomiting medicines is excluded from the line of treatment.

1. To bring out the Thridhosha to its normal physiological activities, laxative are to be administered.
2. Improving haemoglobin content of blood, iron preparations are used.
3. Removal of the causative factors.
4. Pathiam ie diet and other restrictions to normalize the affected thathu and to maintain a longer drug action.

Patients were treated with Agathi keerai kudineer 100 to 200ml at bed time as laxative and Murukkanvithai mathirai (1) with hot water as deworm therapy. Then they were treated with Ilagu Suyamakini Chenduram and Nellikai Legium.

Diet

Siddha system lays a great importance on the observation of rules regarding diet in everyday life because the siddha system has rightly realized that the basic factor of the body is food, that is Annamaya kosam is the first among the five kosams constituting our physical and mental existence to prevent the occurrence of the disease, elaborate inference regarding food item is our daily diet is given in the text books of siddha.

Thirumoolar says

மருந்தே உணவு, உணவே மருந்து

மாறுபாடில்லாத உண்டி மறுத்துண்ணின்

ஊறுபா டில்லையாயு யிர்க்கு

- திருக்குறள்

Herbs, Metals and Minerals used for pandu noi

செப்புகிறேன் பாண்டு காமாலை சோகை

தீர்க்கின்ற சரக்கு வாய்விளங் கந்தாம்

ஒப்பில்லாக் கூவிளை வேர் நத்தைச் சூரி

ஊசிமல்லி மருட்கிழங்கு நல்லவேளை

ஒப்பில்லாமல் மூலிகரிப் பான் சமூலம்

உருக்கு மண்முரே மயம் சுரைக் கொழுந்து

செப்பாம்பித் தளைப்பற்பம் புளியம் பட்டை

சிறு பீளை தாய்க் கொட்டான் சிறுகீரை வேர்
 வேரோடு வெண்காரம் பூனைகக் காலி
 வித்து இளநீர் முத்து கீழக்காய் நெல்லி
 வேரோடு பூவந்திப் பழம் அரத்தை
 வெள்ளைக் காக்கண வேர் காட்டாமணக்கு
 வேரோடு ஜல மஞ்சரிச் செந்துரம்
 வெள்ளாட்டின் நீர் பால் சாரணை வேர்கள்ளி
 நெல்லி வேர் தினைக் கஞ்சி மூசமல்லி
 நீர்முள்ளி சமூலம் பூவரசம் பட்டை
 கொல்லன் கோவை யுடைய சமூகம் வட்டக்
 கிலுகிலுப்பை வாழைநீர் வாழையுட்டி
 வில்வ இலை பாதிரிவேர் குளத்துப் பாலை
 வேரோடு கோமியமும் அஸ்வகந்தி
 கல்யாண சுரப்பான் தக்கோலரும் பிரம்மி
 காரான் திரி கடுகு பாலை குறிஞ்சா
 குறிஞ்சா நாகணம் புளியிலையி னோடு
 கூறிய இவ்வைம்பத் தெட்டு சரக்கும்
 பெருந் தொல்லையாம் பாண்டு சோகை வீக்கம்
 பித்தத் தாலெடுத்த தொரு நோயை யெல்லாம்
 பெருந்த தொலையிலோட்டு விக்கும்
 - வைத்திய சாஸ்திரம்

Diet Regimen for pandu noi

Consuming unbalance and incompatible diet is considered to be the prime causative factor for upsetting the threedosha balance leading to manifestations of various ailments Regarding diet Regimen in vatha pandu the following food items are recommended.

Keerai vagaikal (Greens)

Greens like karisalai, ponnanganni, Arukkeerai, Sirukeerai, Murungai keerai and Manithakkali keerai having haematinic property may be given daily.

Kaikarikal (Vegetables)

Kathiri pinju, Avarai pinju, Murungai pinju, Vazhai kachal may be given along with other food stuffs.

Pazhavakaigal (Fruits)

Pereechai, Orange, Kodimunthiri, Naral, Apple, Atthi are also rich sources for iron which are taken.

Easily digestible foods like Ganjee, Mutton soups, bone soups must be given in acute stages of vatha pandu noi. The goat liver soup is given with orange or apple or grape juice to these patients.

After the normal appetite is restored properly prepared flesh of kadai, kowthari and udumbu can also be give. They tone up the body in the debilitating condition and improve “Raktham” formation.

If oedema is present barley canjee prepared with vellarivithu, mulan vithu and poosunai vithu can be given. This will act as diuretic and reduce oedema.

When the symptoms were improved the patients were advised to practice Yoga and Pranayama according to their physical and mental conditions.

ANEMIA

BLOOD :

Introduction :

Blood is a connective tissue in fluid form.

It is considered as the.

- ❖ Fluid of life because it carries oxygen from the lungs to all parts of the body and carbondioxide from all parts of the body to the lungs.
- ❖ Fluid of growth because it carries nutritive substances from the digestive system and hormones from endocrine glands to all the tissues.
- ❖ Fluid of health because it protects the body against the diseases and gets rid of the waste products and unwanted substances by transporting them to the excretory organs like kidneys.

Properties of blood :

1. Colour :

Arterial blood – Scarlet red.

Venous blood – Purple red.

2. Volume :

Average volume of blood in a normal adult – 5 L.

Volume of blood in a new born baby – 450 ml.

3. Reaction & pH :

Slightly alkaline.

pH \rightarrow 7.4

4. Specific Gravity :

Specific gravity of total blood – 1.052 to 1.061

Specific gravity of blood cells – 1.092 to 1.101

Specific gravity of plasma – 1.022 to 1.026.

5. Viscosity :

Five times more viscous than water.

Composition of blood :

Blood Contains the blood cells which are called formed elements and the liquid portion known as plasma.

1. Blood cells or formed elements :

Three types of cells are present in the blood. They are

- i) Red blood cells or Erythrocytes.
- ii) White blood cells or Leukocytes.
- iii) Platelets or Thrombocytes.

2. Plasma :

Plasma is a straw coloured clear liquid. It contains 91 to 92% of water and 8 to 9% of solids. The solids are the organic and the inorganic substances.

Functions of blood :

1. Nutrient function :

Nutritive substances like glucose amino acids, lipids and vitamins derived from digested food are absorbed from gastrointestinal tract and carried by blood to different parts of the body for growth and production of energy.

2. Respiratory function :

Blood carries oxygen from alveoli of lungs to different tissues and carbon dioxide from tissues to alveoli.

3. Excretory function :

Waste products formed in the tissues during various metabolic activities are removed by blood and carried to the excretory organs like kidney, skin, liver etc, for excretion.

4. Transport of hormones & enzymes :

The hormones, secreted by ductless (endocrine) glands are released directly into the blood. The blood transports these hormones to their target organs / tissues. Blood also transports enzymes.

5. Regulation of water balance :

Water content of the blood is freely interchangeable with interstitial fluid. This helps in the regulation of water content of the body.

6. Regulation of acid base balance :

The plasma proteins and hemoglobin act as buffers and help in regulation of acid base balance.

7. Regulation of body temperature :

Because of the high specific heat of blood, it is responsible for maintaining the thermoregulatory mechanism in the body.

8. Storage function :

Water and some important substances like protein, glucose, sodium and potassium are constantly required by the tissues. Blood serves as a readymade source for these substances.

9. Defensive function :

White blood cells plays important role in the defense of the body.

RED BLOOD CELLS

Introduction :

Red blood cells are also known as erythrocytes (erythros – red).

The red colour of the red blood cell (RBC) is due to the presence of the colouring pigment called hemoglobin.

RBCs play a vital role in transport of respiratory gases.

RBC Count :

	SI units	Non – SI units
Male	$4.5 - 6.5 \times 10^{12}/L$	$4.5 - 6.5 \times 10^6/\text{mm}^3$
Female	$3.8 - 5.8 \times 10^{12}/L$	$3.8 - 5.8 \times 10^6/\text{mm}^3$

Morphology of Red blood cells :

Normal Shape:

Normally, the RBCs are disk shaped and biconcave (dumb bell shaped).

Normal Size:

Diameter : 7.2μ ($6.9 - 7.4\mu$)

Thickness : At the periphery, it is thicker with 2.2μ and

at the centre, it is thinner with 1μ . The difference in thickness is because of the biconcave shape.

Surface area : 120 sq.m.

Volume : 85 – 90 cu.m.

Normal Structure :

RBCs are non – nucleated formed elements in the blood. So, DNA is absent. Mitochondria also are absent and the energy is produced from glycolytic process. Golgi apparatus is also absent in RBC. RBCs do not have insulin receptor and so the glucose uptake by this all is not controlled by insulin.

RBC has a special type of cytoskeleton which is made up of actin and spectrin. Both the proteins are anchored to transmembrane proteins by means of another protein called ankyrin.

PROPERTIES OF RED BLOOD CELLS :

1. Rouleaux formation :

When blood is taken out of the blood vessel, the RBCs pile up one above another like the pile of coins. This property of the RBC is called rouleaux (pleural = rouleau) formation.

2. Specific gravity :

Specific gravity of RBC = 1.092 to 1.101

3. Packed cell volume :

When the blood is collected in a centrifuge tube along with proper anticoagulant and centrifuged for a period of 30 minutes at a speed of 3000 rpm the RBCs settle at the bottom of the tube leaving the clear plasma at the top.

The RBCs form 45% of the total blood. This is called the packed cell volume or hematocrit. The volume of plasma is 55%.

4. Suspension stability :

During circulation, the RBCs remain suspended uniformly in the blood. This property of the RBCs is called the suspension stability.

ERYTHROPOIESIS :

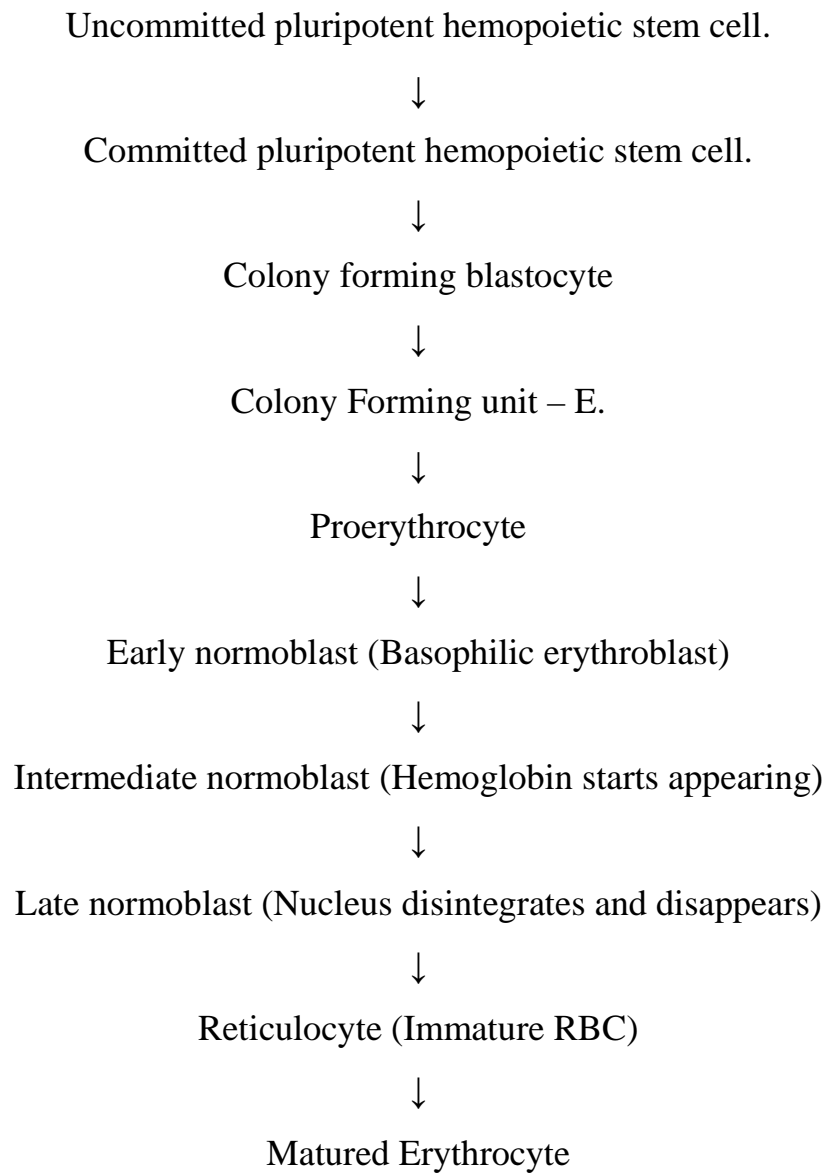
Erythropoiesis is the process which involves the origin, development and maturation of erythrocytes.

Site of Erythropoiesis :

I. Fetal life	Site
1. First 2 months of intrauterine life.	Mesenchyme of yolk sac.
2. From 3 rd month onwards.	Liver, Spleen and Lymphoid organs.
3. During last 3 months of intrauterine life	Red bone marrow and Liver.
II. After birth :	
1. Upto the age of 5 to 6 years	Red bone marrow of all bones.
2. Age 6 yrs to 20 yrs.	Red bone marrow of long bones and all the membranous (flat) bones.
3. After the age of 20 yrs.	From Membranous bones like vertebra, sternum, ribs, scapula, iliac bones and skull bones and from the ends of long bones.

During bone disorders, the RBCs are produced in spleen.

Stages of Erythropoiesis :



It requires 7 days for the development and maturation of RBC from proerythroblast. It requires 5 days upto the stage reticulocyte. The reticulocyte takes 2 more days to become the matured RBC.

Changes during Erythropoiesis :

The stem cells of the colony forming unit – E (CFU – E) pass through different stages and finally become the matured RBCs. During these stages four important changes are noticed.

1. Reduction in size of the cell (from the diameter of 25μ to 7.2μ)
2. Disappearance of nucleoli and nucleus
3. Appearance of hemoglobin.
4. Change in the staining properties of the cytoplasm.

Factors necessary for Erythropoiesis :

Development and maturation of erythrocytes require variety of factors which are classified into 3 categories:

- I) General factors.
- II) Maturation factors.
- III) Factors necessary for hemoglobin formation.

I. General Factors:

General factors necessary for erythropoiesis are :

1. Erythropoietin.
2. Thyroxine.
3. Hemopoietic growth factors
4. Vitamins.

1. Erythropoietin :

The most important general factor for erythropoiesis is the hormone erythropoietin. It is also called homopoietin or erythrocyte stimulating factor.

Erythropoietin is a glycoprotein secreted by peritubular capillaries of kidney.

Hypoxia is the stimulant for the secretion of erythropoietin.

Erythropoietin promotes the following processes:

- i) Production of proerythroblasts from the stem cells in CFU – E of the bone marrow.
- ii) Development of proerythroblasts into matured RBCs through the normoblastic stages.

- iii) Release of matured erythrocytes into blood. Even some reticulocytes (immature erythrocytes) are released along with matured RBCs.

After secretion, it takes 4 to 5 days to show the action.

2. **Thyroxine :**

Being a general metabolic hormone, thyroxine accelerates the process of erythropoiesis at many levels.

3. Hemopoietic growth factors :

Hemopoietic growth factors or growth inducers are the interleukins and stem cell factor (Steel factor). Generally, these factors induce the proliferation of pluripotent stem cells. Interleukins involved in erythropoiesis are IL – 3, IL -6 and IL -11.

4. Vitamins :

The vitamins, which are necessary for erythropoiesis are :

Vitamin B,

Vitamin C,

Vitamin D and

Vitamin E.

II. Maturation factors:

Vitamin B₁₂, intrinsic factor and folic acid are necessary for the maturation of RBCs.

1. Vitamin B₁₂ (Cyanocobalamin):

This is essential for maturation of erythrocytes. The deficiency of vitamin B₁₂ causes pernicious anaemia. So vitamin B₁₂ is called antipernicious factor.

Source of vit. B₁₂ :

Vit. B₁₂ is called extrinsic factor because it is obtained mostly from diet. Its absorption from the intestine requires the presence of intrinsic factor of castle. Vit. B₁₂ is stored mostly in the liver and in small quantity in muscle. When necessary, it is transported to the bone marrow to promote maturation of RBCs. It is also produced in the large intestine by the intestinal flora.

Action of vit. B₁₂ :

Vit. B₁₂ is essential for synthesis of DNA. Its deficiency leads to failure in maturation of the cell and reduction in the cell division. Also, the cells are larger with fragile and weak cell membrane.

2. Intrinsic Factor of castle :

It is produced in gastric mucosa and it is essential for the absorption of Vit. B₁₂ from intestine into the blood. In the absence of intrinsic factor, Vit. B₁₂ is not absorbed. This happens in severe gastritis, ulcers and gastrectomy. The deficiency of intrinsic factor also causes pernicious anemia since the Vit. B₁₂ is not absorbed. The extrinsic and intrinsic factors are together called hematinic principle.

3. Folic acid :

Folic acid is also essential for maturation. It is required for the synthesis of DNA. In the absence of folic acid, the synthesis of DNA decreases causing failure of maturation. This leads to anemia in which, the cells are larger and appear in megaloblastic (Proerythroblastic) stage. Anemia due to folic acid deficiency is called megaloblastic anemia.

III. Factors necessary for hemoglobin formation :

Various materials are essential for the formation of hemoglobin in the RBCs. The deficiency of these substances decreases the production of hemoglobin leading to anemia. Such factors are :

1. First class proteins and amino acids :

Proteins of high biological value are essential for the formation of hemoglobin. Amino acids derived from these proteins are required for the synthesis of protein part of hemoglobin, the globin.

2. Iron :

It is necessary for the formation of heme part of the hemoglobin.

3. Copper :

It is necessary for the absorption of iron from the gastrointestinal tract.

4. Cobalt and nickel :

Cobalt and nickel are essential for the utilization of iron during hemoglobin formation.

5. Vitamins :

Vit. C, Riboflavin, Nicotinic acid and pyridoxine are also essential for the formation of Hemoglobin.

VARIATIONS IN SIZE OF RED BLOOD CELLS :

Under physiological conditions, the size of RBC in venous blood is slightly larger than those in arterial blood.

In pathological conditions, the variations in size of RBCs are :

1. Microcytes – decrease in size.
2. Macrocytes – increase in size.

3. Anisocytes – cells without uniform size.

Microcytes :

Microcytes are present in :

- i) Iron deficiency anaemia.
- ii) Prolonged forced breathing.
- iii) Increased osmotic pressure in blood.

Macrocytes :

Macrocytes are present in

- i) Megaloblastic anemia.
- ii) Muscular exercise.
- iii) Decreased osmotic pressure in blood.

Anisocytes :

Anisocytes occurs in pernicious anemia.

VARIATIONS IN SHAPE OF RBCs:

The shape of RBCs is altered in many conditions including different types of anemia.

1. Genation :

Shrinkage as in hypertonic solution.

2. Spherocytosis :

Globular form as in hypotonic solution.

3. Elliptocytosis :

Elliptical shape as in certain types of anemia.

4. Sicke cell :

Crescentic shape as in sickle cell anemia.

5. Poikilocytosis :

Unequal shapes due to deformed cell membrane. The shape will be of flask hammer or any other unusual shape.

VARIATIONS IN STRUCTURE OF RBCs :

1. Punctate Basophilism :

The striated appearance of RBCs by the presence of dots of basophilic materials (porphyrin) is called punctate basophilism. It occurs in conditions like lead poisoning.

2. Ring :

Ring or twisted strands of basophilic material appear in the periphery of the RBCs. This is also called the goblet ring. This appears in the RBCs in certain types of anemia.

3. Howell – Jolly Bodies :

In certain types of anemia, some nuclear fragments are present in the ectoplasm of the RBCs. These nuclear fragments are called howell – Jolly bodies.

LIFESPAN AND FATE OF RBCs :

Average lifespan of RBC is about 120 days. After the lifetime the senile (old) RBCs are destroyed in reticuloendothelial system.

When the cells become older (120 days) the cell membrane becomes more fragile. The diameter of the capillaries is less or equal to that of RBC. The younger RBCs can pass through the capillaries easily. However, because of the fragile nature, the older cells are destroyed while trying to squeeze through the capillaries. The destruction occurs mainly in the capillaries of spleen, because the diameter of splenic capillaries is very small. So, the spleen is called graveyard of RBCs.

The destroyed RBCs are fragmented. From the fragmented parts, the hemoglobin is released and degraded into iron, globin and porphyrin. Iron combines with the protein apoferritin to form ferritin, which is stored in body. Globin enters the protein depot. The porphyrin is degraded and finally forms bilirubin which is excreted by liver through bile.

Daily 10% RBCs, which are senile, are destroyed in normal young healthy adults. It causes release of about 0.6g% of hemoglobin into the plasma. From this, 0.9 to 1.5 mg% bilirubin is formed.

FUNCTIONS OF RBCs :

1. Transport of oxygen from the lungs to the tissues :

Hemoglobin in RBC combines with oxygen to form oxyhemoglobin. About 97% of oxygen is transported in blood in the form of oxyhemoglobin.

2. Transport of carbon dioxide from the tissues to the lungs :

Hemoglobin combines with carbondioxide and form carboxyhaemoglobin. About 30% of carbon dioxide is transported in this form.

RBCs contain a large amount of the carbonic anhydrase. This enzyme is necessary for the formation of bicarbonate from water and carbon dioxide. Thus it helps to transport carbon dioxide in the form of bicarbonate from tissues to lungs. About 63% of carbon dioxide is transported in this form.

3. Buffering action in blood :

Hemoglobin functions as a good buffer. By this action, it regulates the hydrogen ion concentration and thereby plays a role in the maintenance of acid base balance.

4. In Blood group determination :

RBCs carry the blood group antigens like A agglutinin, B agglutinin and Rh factor. This helps in determination of blood group and enables to prevent reactions due to incompatible blood transfusion.

HEMOGLOBIN (Hb)

Introduction :

Hemoglobin is the red blood pigment, exclusively found in erythrocytes. It is a chromoprotein forming 95% of dry weight of RBC and 30 to 34% of wet weight. The molecular weight of hemoglobin is 68,000. It performs two important biological functions concerned with respiration:

- i) Delivery of O₂ from the lungs to the tissues.
- ii) Transport of CO₂ and protons from tissues to lungs for excretion.

It also acts as a buffer.

NORMAL Hb LEVELS:

Average hemoglobin (Hb) content in blood is 14 to 16g%. However, the value varies depending upon the age and sex of the individual.

Age :

At birth	:	25g%
After 3 rd month	:	20g%
After 1 Year	:	17g%
From puberty onwards	:	14 to 16g%

At the time of birth, Hb content is very high because of increased RBC count.

	Hb level	Hb level
Sex :	In ST unit	in non – ST unit.
In adult males	: 130 – 180 g/l	13 – 18 g/dl
In adult females	: 115 – 165 g/l	11.5 – 16.5 g/dl

STRUCTURE :

Hb is a conjugated protein. It consists of a protein combined with an iron containing pigment. The protein part is globin and the iron containing pigment is heme. Hence also forms a part of structure of myoglobin (oxygen binding pigment in muscles)

Iron :

Normally, it is present in ferrous (Fe^{++}) form. It is an unstable or loose form in some abnormal conditions, the iron is converted into ferric (Fe^{+++}) state, which is a stable form.

Porphyrin :

The pigment part is called porphyrin it is formed by four pyrrole rings (tetrapyrrole) called I, II, III and IV. The pyrrole rings are attached to one another by methane (CH_4) bridges.

The iron is attached to N- of each pyrrole ring and N- of globin molecule.

Globin :

This contains four polypeptide chains. Among the four polypeptide chains, two are α – chains and two are β Chains.

(i) α Chain :

Molecular weight – 15, 126.

No. of amino acids – 141.

(ii) β Chain :

Molecular weight - 15, 866

No. of amino acids – 146.

NORMAL MAJOR TYPES OF HEMOGLOBINS:

Type	Composition and symbol	Percentage of total Hb
Hb A1	$\alpha_2 \beta_2$	90%
Hb A2	$\alpha_2 \delta_2$	< 5%
Hb F	$\alpha_2 \gamma_2$	< 2%
Hb A _{1c}	$\alpha_2 \beta_2$ glucose.	< 5.4%

SYNTHESIS OF Hb:

Synthesis of Hb actually starts in proerythroblastic stage. However, Hb appears in the intermediate normoblastic stage only. The production of

the hemoglobin is continued until the stage of reticulocyte. The heme portion of hemoglobin is synthesized in mitochondria from acetic acid and the glycine. The sequence of events in the synthesis of hemoglobin:

1. During krebs cycle, the acetic acid is converted into succinyl CoA
2. Two molecules of succinyl CoA combine with two molecules of glycine to form pyrrole compound.
3. Four pyrrole compounds combine to form protoporphyrin.
4. Protoporphyrin is of many types. Only protoporphyrin IX is involved in the formation of heme molecule by combining with iron.
5. Each heme molecule combines with one globin molecule to form hemoglobin.

The protein part, globin is synthesized in ribosomes.

DESTRUCTION OF Hb:

After the lifespan of 120 days, the RBC is destroyed in the reticuloendothelial system particularly in spleen and the hemoglobin is released into plasma. Soon, the hemoglobin is degraded in the reticuloendothelial cells and split into globin, iron and porphyrin.

Globin is utilized for the resynthesis of Hb. Iron is stored in the body as ferritin and hemosiderin, which are reutilized for synthesis for new Hb.

Porphyrin is converted into a green pigment called biliverdin. In human being, most of the biliverdin is converted into a yellow pigment called bilirubin. Bilirubin and biliverdin are together called the bile pigments.

FUNCTIONS :

1. Transport of respiratory gases:

The main function of hemoglobin is the transport of respiratory gases:

- (i) Oxygen from the lungs to tissues.
- (ii) Carbon dioxide from tissues to lungs

(i) Transport of oxygen :

When oxygen binds with Hb, a physical process called oxygenation occurs resulting in the formation of oxyhemoglobin. The iron remains in ferrous state in this compound. Oxyhemoglobin is an unstable compound and the combination is reversible, ie. The oxygen can be released from this compound.

When oxygen is released from oxyhemoglobin, it is called reduced hemoglobin or ferrohemoglobin.

(ii) Transport of carbon dioxide :

When carbon dioxide binds with hemoglobin, carbohemoglobin is formed. It is also an unstable compound and the combination is reversible, ie. the carbondioxide can be released from this compound. The affinity of Hb for carbondioxide is 20 times more than for oxygen.

2. Buffer action :

Hb acts as a buffer and plays an important role in acid base balance.

IRON

Introduction :

The total content of iron in an adult body is 3 – 5 g. about 70% of this occurs in the erythrocytes of blood as a constituent of Hb. Hence is the most predominant iron containing substance. It is a constituent of several proteins / enzymes (hemoproteins) – hemoglobin, myoglobin, cytochromes, xanthine oxidase, catalase, tryptophan pyrrolase peroxidase, certain other proteins contain non heme hence iron eg. Transferrin. Ferritin, hemosiderin.

Distribution of iron in the body:

The approximate distribution of iron in the body is as follows:

In the hemoglobin	:	65 to 68%
In the muscle as myoglobin	:	4%

As intracellular oxidative heme compound	:	1%
In the plasma as transferrin	:	0.1%
Stored in the reticuloendothelial system	:	25 to 30%

Dietary requirements :

Adult man	-	10 mg / day
Menstruating woman	-	18 mg / day.
Pregnant and lactating women	-	40 mg / day

Sources :

Dietary iron is available in two forms called heme and non - heme.

Heme iron is present in fish, meat and chicken. Non-heme iron is available in vegetables, grains and cereals.

Rich Sources	-	Organ meats (liver, heart, kidney)
Good Sources	-	Leafy vegetables, pulses, Cereals, fish, apples, Dried fruits, molasses.
Poor Sources	-	Milk, wheat, polished rice.

Absorption of iron :

Iron is mainly absorbed from the small intestine. It is absorbed through the intestinal cells by pinocytosis and transported into the plasma. Bile is essential for the absorption of iron.

Transport and storage of iron :

Immediately, after absorption into the blood, iron combines with a β – globulin called apotransferrin to form transferrin and is transported in this form in the plasma. Iron combines loosely with the globin and can be released easily at any region of the body.

Iron is stored in large quantities in the reticuloendothelial cells and liver hepatocytes. In other cells also, it is stored in small quantities. In the cytoplasm of the cell, iron is stored as ferritin in large amount. Small quantity of iron is also stored as hemosiderin.

Factors affecting iron absorption :

1. Acidity, ascorbic acid and cysteine promote iron absorption.
2. In iron deficiency anemia, Fe absorption is increased to 2-10 times that of normal.
3. Small peptides and amino acids favour iron uptake.

4. Phytate (found in cereals) and oxalate (found in leafy vegetables) interfere with Fe absorption.
5. A diet with high phosphate content decreases Fe absorption while low phosphate promotes.
6. Impaired absorption of iron is observed in malabsorption syndromes such as steatorrhea.
7. In patients with partial or total surgical removal of stomach and / or intestine, iron absorption is severely impaired.

Daily loss of iron :

In males, about 1 mg of iron is excreted everyday through feces. In females, the amount of iron loss is very much high. This is because of the menstruation.

1g of hemoglobin contains 3.34 mg of iron. Normally 100ml of blood contains 15g of hemoglobin and about 50mg of iron. So, if 100ml of blood is lost from the body, there is a loss of about 50mg of iron. In females, during every menstrual cycle, about 50 ml of blood is lost by which 25 mg of iron is lost. That is why, the iron content is always less in females than in males.

Iron is lost during hemorrhage and blood donation also. If 450ml of blood is donated, about 225mg of iron is lost.

Regulation of total body iron :

Absorption and excretion of iron are maintained almost equally under normal physiological conditions. When the iron storage is saturated in the body, it automatically reduces the further absorption of iron from the gastrointestinal tract by feedback mechanism.

The factors which reduce the absorption of iron are :

1. Stoppage of apotransferrin formation in the liver, so that, the iron could not be absorbed from the intestine.
2. Reduction in the release of iron from the transferrin so that, transferrin is completely saturated with iron and further absorption is prevented.

Functions :

1. Iron mainly exerts its functions through the compounds in which it is present.
 - ❖ Hemoglobin and myoglobin are required for the transport of O₂ and CO₂.
 - ❖ Cytochromes and certain non-heme proteins are necessary for electron transport chain and oxidative phosphorylation.
 - ❖ Peroxidase, the lysosomal enzyme, is required for phagocytosis and killing of bacteria by neutrophils.
2. Iron is associated with effective immunocompetence of the body.

PACKED CELL VOLUME AND BLOOD INDICES

Packed cell volume :

Packed cell volume (PCV) is the hematocrit value expressed as the percentage of cellular elements with that of whole blood. Or it is the measure or proportion of blood volume that is occupied by RBCs.

Normal values of PCV or Hematocrit :

	In SI units.	In Non SI units
Males	0.40 – 0.54	40 – 54%
Females	0.37 – 0.47	37 – 47%

BLOOD INDICES :

The various blood indices are,

1. Mean corpuscular volume (MCV)
2. Mean corpuscular hemoglobin (MCH)
3. Mean corpuscular hemoglobin concentration (MCHC)
4. Colour index.

1. Mean corpuscular volume (MCV) :

MCV is the average volume of a single RBC and it is expressed in cubic micron (cu.μ)

$$\text{MCV} = \frac{\text{Volume of packed cells in ml per 1000 ml of blood}}{\text{RBCs in million per cu. mm. of blood}}$$

Normal value :

	In SI units	In non SI units
MCV	78 – 98 fl	78 – 98 cu.μ.

MCV is more in pernicious anemia and megaloblastic anemia in which the RBCs are macrocytic in nature. MCV is less in microcytic anemia.

2. Mean corpuscular Hemoglobin (MCH) :

It is the quantity or amount of Hb present in one RBC. It is expressed in microgram or picogram (pg).

$$\text{MCH} = \frac{\text{Hb in grams per 1000 ml of blood}}{\text{RBC count in million / cu. mm}}$$

Normal value :

$$\text{MCH} = 27 - 32 \text{ pg.}$$

It decreases or remains normal in pernicious anemia and megaloblastic anemia, in which RBCs are macrocytic and normochromic or hypochromic. It decreases in hypochromic anemia. When MCH is normal, it is called normochromic state.

3. Mean corpuscular Hemoglobin concentration (MCHC) :

This is the concentration of Hb in one RBC. It is the amount of Hb expressed in relation to the volume of one RBC. So, its unit is percentage.

Hb in grams / 100ml of blood

$$\text{MCHC} = \frac{\text{Hb in grams / 100ml of blood}}{\text{PCV in 100ml of blood}} \times 100$$

PCV in 100ml of blood

Normal Value :

$$\text{MCHC} = 30 \text{ to } 38\%$$

This is the most important absolute value in the diagnosis of anemia. It decrease in iron deficiency anemia in which RBCs are microcytic and hypochromic.

4. Color Index (CI) :

This is the ratio between the percentage of Hb and the percentage of RBCs in the blood. Actually, it is the average Hb content in one cell of a patient compared to average Hb content in one cell of a normal person.

$$\text{Color index} = \frac{\text{Hemoglobin \%}}{\text{RBC \%}}$$

$$\text{Where Hb \%} = \frac{\text{Hb content in the subject}}{\text{Normal Hb content}} \times 100$$

$$\text{RBC \%} = \frac{\text{RBC count in the subject}}{\text{Normal RBC count}} \times 100$$

Normal Value :

$$\text{CI} = 0.8 \text{ to } 1.2.$$

It raises in pernicious anemia and megaloblastic anemia. It is reduced in iron deficiency anemia. And, it is normal in normocytic normochromic anemia

ANEMIA

Introduction :

Anemia is defined as a hemoglobin concentration in blood below the lower limit of the normal range for the age and sex of the individual in adults, the lower extreme of the normal hemoglobin is taken as 13.0g / dl for males and 11.5g/dl for females. Newborn infants have higher hemoglobin level and, therefore, 15g/dl is taken as the lower limit at birth, whereas at 3 months the lower level is 9.5g/dl. Although hemoglobin value is employed as the major parameter for determining whether or not anemia is present, the red cell counts, hematocrit (PCV) and absolute values (MCV, MCH and MCHC) provide alternate means of assessing anemia.

Generally, anemia occurs because of :

1. Decreased production of RBC.
2. Increased destruction of RBC or
3. Excess loss of blood from the body.

All these incidents are caused either by inherited disorders or environmental influences such as nutritional problem, infection and exposure to drugs or toxins.

Pathophysiology of Anemia :

Subnormal level of Hb causes lowered oxygen – carrying capacity of the blood. This, in turn, initiates compensatory physiologic adaptations such as :

- ❖ Increased release of oxygen from Hb :
- ❖ Increased blood flow to the tissues ;
- ❖ Maintenance of the blood volume ; and
- ❖ Redistribution of blood flow to maintain the cerebral blood supply.

Eventually, however, tissue hypoxia develops causing impaired functions of the affected tissues. The degree of functional impairment of individual tissues is variable depending upon their oxygen requirements. Tissues with high oxygen requirement such as the heart, CNS and the skeletal muscle during exercise, bear the clinical effects of anemia.

Clinical features of Anemia :

The Hb level at which symptoms and signs of anemia develop depends upon 4 main factors.

1. The speed of onset of anemia :

Rapidly progressive anemia causes more symptoms than anemia of slow onset as there is less time for physiologic adaptation.

2. The severity of anemia :

Mild anemia produces no symptoms or signs but a rapidly developing severe anemia (Hb below 6g/dl) may produce significant clinical features.

3. The age of the patient :

The young patients due to good cardiovascular compensation tolerate anemia quite well as compared to the elderly. The elderly patients develop cardiac and cerebral symptoms more prominently due to associated cardiovascular disease.

4. The hemoglobin dissociation curve :

In anemia, the affinity of Hb for oxygen is depressed as 2, 3 – BPG in the red cells increase. As a result, oxyhemoglobin is dissociated more readily to release free oxygen for cellular use, causing a shift of the oxyhemoglobin dissociation curve to the right.

In older patients, there may be symptoms of.

Cardiac failure.

Angina Pectoris,

Intermittent claudication,

Confusion and

Visual disturbances

Signs :

A few general signs common to all types of anemias are as under :

1. Pallor :

Pallor is the most common and characteristic sign which may be seen in the mucous membrane, conjunctivae and skin.

2. Cardiovascular system :

A hyperdynamic circulation may be present with tachycardia, collapsing pulse, cardiomegaly, midsystolic flow murmur, dyspnoea on exertion, and in the case of elderly, congestive heart failure.

3. Central nervous system :

The older patients may develop symptoms referable to the CNS such as attack of faintness, giddiness, headache tinnitus, drowsiness, numbness and tingling sensations of the hands and feet, dizziness or vertigo especially when standing and increased sensitivity to cold.

4. Respiratory system :

There is increase in rate and force of respiration. Sometime, it leads to breathlessness and dyspnoea. Oxygen hemoglobin dissociation curve is shifted to right.

5. Ocular manifestations :

Retinal hemorrhages may occur if there is associated vascular disease or bleeding diathesis.

6. Reproductive system :

Menstrual disturbances such as amenorrhoea and menorrhagia and loss of libido are some of the manifestations involving the reproductive system in anemic subjects.

7. Renal system :

Mild proteinuria and impaired concentrating capacity of the kidney may occur in severe anemia.

8. Gastrointestinal system :

Anorexia, flatulence, nausea, constipation and weight loss may occur.

9. Metabolism :

Basal metabolic rate increases in severe anemia.

10. Muscles :

Muscles become weak and the patient feels lack of energy and fatigued quite often and quite easily.

11. Nails & Hair :

Pallor can be seen in nail bed. The nails become brittle and easily breakable. Thinning, loss and early grayness of hair occur.

Classification of anemia :

Anemia is classified by 2 methods :

- a. Morphological classification.
- b. Etiological classification.

A. Morphological classification :

Based on the red cell size, hemoglobin content and red cell indices, anemias are classified into 4 types :

1. Normocytic normochromic anaemia :

The size and Hb content of RBC are normal. But the number of RBC is less. MCV, MCH, MCHC are all normal.

eg. After acute blood loss.

Hemolytic anemias,

Bone marrow failure,

Anemia of chronic disorders.

2. Macrocytic Normochromic Anemia :

The RBCs are large in size with normal Hb content. The RBC count decreases.

3. Macrocytic hypochromic Anemia :

The RBCs are larger in size. The hemoglobin content in the cell (MCH) is less so the cells are pale.

In macrocytic anemias, MCV is raised.

eg: Megaloblastic anemia due to deficiency of Vit B12 or folic acid.

4. Microcytic hypochromic Anemia :

The RBCs are smaller in size.

MCV, MCH, MCHC are all reduced.

eg: Iron deficiency anemia.

Sideroblastic anemia

Thalassemia.

Anemia of chronic disorders.

B. ETIOLOGICAL CLASSIFICATION :

On the basis of the etiology (cause of the disease), the anemia is divided into 5 types :

1. Hemorrhagic anemia.
2. Hemolytic anemia.
3. Nutrition deficiency anemia.
4. Aplastic anemia.
5. Anemia of chronic disease.

(1) Hemorrhagic anemia :

Anemia due to hemorrhage is known as hemorrhagic anemia. It occurs both in acute and chronic hemorrhagic conditions. Hemorrhage occurs in conditions like accident. Ulcer, excessive uterine bleeding, purpura and hemophilia.

Acute hemorrhage :

It refers to sudden loss of a large quantity of blood as in the case of accident. Within about 24 hours after the hemorrhage, the plasma portion of blood is replaced. However, the replacement of RBCs does not occur quickly and it takes at least 4-6 weeks. So with less number of RBCs, hemodilution occurs.

However, morphologically the RBCs are normocytic, normochromic.

Decreased RBC count causes hypoxia which stimulates the bone marrow to produce more number of RBC so, the condition is corrected within 4 – 6 weeks.

Chronic hemorrhage :

It refers to loss of blood by internal or external bleeding over a long period of time.

It occurs in conditions like peptic ulcer, purpura, hemophilia and menorrhagia.

Due to continuous loss of blood, lot of iron is lost from the body causing iron deficiency. This affects the synthesis of Hb resulting in less Hb content in the cells. The cells also become small. Hence, the RBCs are microcytic and hypochromic.

(2) HEMOLYTIC ANEMIA :

Hemolysis means destruction of RBCs hemolytic anemia occurs because of excess destruction of RBCs.

Causes of excess hemolysis are :

Liver failure

Renal disorder.

Hypersplenism.

Burns.

Infections like malaria and septicemia.

Poisoning by chemical substances like lead, coal and tar.

Presence of isoagglutinins like anti Rh

Congenital or acquired default in the shape of RBCs.

When the shape is abnormal, RBCs become more fragile and hemolysis occurs easily. It occurs in two inherited conditions called sickle cell anemia and thalassemia.

(i) Sick cell anemia :

It is a congenital anomaly and found mostly in blacks. It is due to the abnormal hemoglobin called hemoglobin S (normal adult Hb is Hb A). In this, α chains are normal and β chains are abnormal. The molecules of Hb S polymerize into long chains and precipitate inside the cells. Because of this, RBCs attain sickle (crescent) shape and become more fragile leading to hemolysis.

In children, hemolyzed sickle cells aggregate and block the blood vessels leading to infarction. The infarction is common in small bones. The infarcted small bones in hand and foot results in varying length in the digits. This condition is known as hand and foot syndrome. Jaundice also occurs in these children.

Clinical features :

The clinical manifestations of homozygous sickle cell disease are widespread. The symptoms begin to appear after 6th month of life when most of the HbF is replaced by HbS. Infection and folic acid deficiency result in more severe clinical manifestations. These features are as under :

1. Anemia :

There is usually severe chronic hemolytic anemia (primarily extravascular) with onset of aplastic crisis in between, the symptoms of anemia are generally mild since Hbs gives up oxygen more readily than HbA to the tissues.

2. Vaso – occlusive phenomena :

Patients of SS develop recurrent vaso – occlusive episodes throughout their lives due to destruction of capillary blood flow by sickled red cells upon deoxygenation or dehydration. Vasoobstruction affecting different organs and tissues results in infarcts which may be of 2 types :

(a) Microinfarcts affecting particularly the abdomen, chest, back and joints and are the cause of recurrent painful crises in SS.

(b) Macroinfarcts involving most commonly the spleen (the splenic sequestration, autosplenectomy), bone marrow (pains), bones (aseptic necrosis, osteomyelitis), lungs (pulmonary infections), kidneys (renal cortical necrosis), CNS (stroke), retina (damage) and skin (ulcers), and result in anatomic and functional damage to these organs.

3. Constitutional symptoms :

In addition to the features of anemia and infarction, patients with SS have impaired growth and development and increased susceptibility to infection due to markedly impaired splenic function.

(ii) THALASSEMIA :

The thalassemias are a diverse group of hereditary disorders in which there is reduced rate of synthesis of one or more of the globin polypeptide chains. Thalassemias are first described in people of mediterranean countries (North Africa, Southern Europe) from where it derives its name 'mediterranean anemia'. The word 'thalasse' in Greek means 'the sea' since the condition was found commonly in regions around the Mediteranean basin. It also occurs in the middle east India, south – east Asia and in general, in blacks.

Normally, an individual inherits 2 β - globin genes located one each on two chromosomes 11, and 2 α - globin genes one each on 2 chromosomes 16, from each parent ie. normal adult Hb is $\alpha_2 \beta_2$ (Hb A). Depending upon the whether the genetic defect or deletion lies in transmission of α - or β - globin chain genes, thalassemias are classified into α - and β - thalassemias.

a) α - Thalassemia :

α - Thalassemias are disorders in which there is defective synthesis of globin chains resulting in depressed production of hemoglobins that contain chains (ie) HbA, HbA₂ and HbF. The α - thalassemias are most commonly due to deletion of one or more of the α - chain genes. Depending upon the no. of genes deleted, α thalassemias are classified into 4 types.

1. Four α - gene deletion – Hb Bart's Hydrops foetalis.
2. Three α - gene deletion – HbH disease.
3. Two α - gene deletion - α - thalassemia trait.
4. One α - gene deletion - α - thalassemia trait (carrier).

Clinical features :

Hb Bart's Hydrops foetalis is incompatible with life due to severe tissue hypoxia. The condition is either fatal in utero or the infant dies shortly after birth of born alive, the features of severe Rh hemolytic disease are present.

HbH Disease :

HbH Disease is generally present as a well – compensated hemolytic anemia. The features are intermediate between that of β - thalassemia minor and major. The severity of anemia fluctuates and may fall to very low levels

during pregnancy or infections. Majority of patients have splenomegaly and may develop cholelithiasis.

α -Thalassemia trait :

α -thalassemia trait due to two α - chain gene deletion is asymptomatic. It is suspected in a patient of refractory microcytic hypochromic anemia in whom iron deficiency and β - thalassemia minor have been excluded and the patient belongs to the high – risk ethnic group.

α -Thalassemia trait (carrier) :

One gene deletion α - thalassemia trait is a silent carrier state.

6) β - Thalassemia :

The β - thalassemias are caused by decreased rate of β - chain synthesis resulting in reduced formation of HbA in the red cells. The molecular pathogenesis of the β - thalassemias is more complex. In contrast to α - thalassemias, gene deletions rarely ever cause β - thalassemia and is only seen in an entity called Hereditary persistence of foetal Hemoglobin (HPFH). Instead, most of β - thalassemias arise from different types of mutations of β - globin gene resulting from single base changes. The symbol β^0 is used to indicate the complete absence of synthesis while β^+ denotes partial synthesis of the β - globin chains.

Depending upon the extent of reduction in β – chain synthesis, there are 3 types of β - thalassemia :

1. Homozygous form :

β – thalassemia major mediterranean or cooley's anemia. It is the most severe form of congenital hemolytic anemia. It is further of 2 types.

a) β^0 thalassemia major characterised by complete absence of β - chain synthesis.

b) β^+ thalassemia major having incomplete suppression of β - chain synthesis.

2. β - thalassemia intermedia :

It is β - thalassemia of intermediate degree of severity that does not require regular blood transfusions. These cases are genetically heterozygous (β^0 / β or β^+ / β).

3. Heterozygous form : β - thalassemia minor (trait) :

It is a mild asymptomatic condition in which there is moderate suppression of β - chain synthesis.

Clinical features :

1. β - thalassemia major :

Clinical manifestations appear insidiously and are as under.

a) Anemia starts appearing within the first 4 - 6 months of life when the switch over from α - chain to β - chain production occurs :

b) Marked hepatosplenomegaly occurs due to excessive red cell destruction, extramedullary hematopoiesis and iron overload.

c) Expansion of bones occurs due to marked erythroid hyperplasia leading to thalassemic facies and malocclusion of the jaw.

d) Iron overload due to repeated blood transfusions causes damage to the endocrine organs resulting in slow rate of growth and development, delayed puberty, diabetes mellitus and damage to the liver and heart.

2. β - Thalassemia. Minor :

Clinically, the condition is usually asymptomatic and the diagnosis is generally made when the patient is being investigated for a mild chronic anemia. The spleen may be palpable.

3. NUTRITION DEFICIENCY ANEMIA :

Nutritive substances such as iron, proteins and vitamins like C, B₁₂ and folic acid are necessary for erythropoiesis. The deficiency of these substances leads to nutrition (nutritional) deficiency anemia.

(i) IRON DEFICIENCY ANEMIA :

Iron deficiency anemia is the most common type of anemia. It develops due to inadequate availability of iron for Hb synthesis. The RBCs are microcytic and hypochromic. Causes of iron deficiency anemia are :

Loss of blood.

Decreased intake of iron.

Poor absorption of iron from intestine.

Increased demand for iron in conditions like growth and pregnancy.

Clinical features :

Iron deficiency anemia is much more common in women between the age of 20 and 45 yrs than in men; at periods of active growth in infancy, childhood and adolescence, and is also more frequent in premature infants. Initially, there are usually no clinical abnormalities. But subsequently, in addition to features of the underlying disorder causing the anemia, the clinical consequences of iron deficiency manifest in two ways – anemia itself & epithelial tissue changes.

a) Anemia :

The onset of iron deficiency anemia is generally slow. The usual symptoms are of weakness, fatigue, dyspnoea on exertion, palpitations and

pallor of the skin, mucous membranes and sclerae. Older patients may develop angina and congestive cardiac failure. Patients may have unusual dietary cravings such as PICA. Menorrhagia is a common symptom in iron deficient women.

b) Epithelial tissue changes :

Long – standing chronic iron deficiency anemia causes epithelial tissue changes in some patients. The changes occur in the nails (Koilonychia or spoon – shaped nails), tongue (atrophic glossitis), mouth (angular stomatitis), and oesophagus causing dysphagia from development of thin, membranous webs at the postcricoid area (Plummer – Wilson syndrome)

(ii) PROTEIN DEFICIENCY ANEMIA ;

Due to deficiency of proteins, the synthesis of hemoglobin is reduced. The RBCs are macrocytic and hypochromic.

(iii) PERNICIOUS ANEMIA (or) ADDISON'S ANEMIA :

It is due to atrophy of the gastric mucosa because of autoimmune destruction of parietal cells. The gastric atrophy results in decreased production of intrinsic factor and poor absorption of vitamin B₁₂ which is the

maturation factor for RBC. The RBCs are larger and immature with almost normal hemoglobin content. So, cells are macrocytic and normochronic.

It was very difficult to treat the patients and the disease was considered to be fatal. So, it was called pernicious anemia. The synthesis of Hb is almost normal in this type of anemia.

Pernicious anemia is common in old age and it is more common in females than in males. It is associated with other autoimmune diseases like disorders of thyroid gland, Addison's disease etc.,

Clinical Features :

The disease has insidious onset and progresses slowly. The clinical manifestations are mainly due to vitamin B₁₂ deficiency. These include : lemon yellow colour of skin (due to anemic paleness and mild jaundice), anaemia, glossitis, neurological abnormalities (neuropathy, subacute combined degeneration of the spinal cord, retrobulbar neuritis), gastrointestinal manifestations (diarrhoea, anorexia, weight loss, dyspepsia), hepatosplenomegaly, congestive heart failure and hemorrhagic manifestations.

(iv) MEGALOBLASTIC ANEMIA :

The megaloblastic anemias are disorders caused by impaired DNA synthesis and are characterised by distinctive abnormality in the

hematopoietic precursors in the bone marrow in which the maturation of the nucleus is delayed relative to that of the cytoplasm. Since cell division is slow but cytoplasmic development progress normally, the nucleated red cell precursors tend to be larger which Ehrlich in 1880 termed megaloblasts. Megaloblasts are both morphologically and functionally abnormal with the result that the mature red cells formed from them and released into the peripheral blood are also abnormal in shape and size, the most prominent abnormality being macrocytosis.

The underlying defect for the asynchronous maturation of the nucleus is defective DNA synthesis due to deficiency of vitamin B₁₂ (cobalamin) and / or folic acid (folate)

Clinical features :

Deficiency of Vit B₁₂ and folate may cause the following clinical manifestations which may be present singly or in combination and in varying severity.

1. Anemia :

Macrocytic megaloblastic anemia is the cardinal feature of deficiency of Vit. B₁₂ and / or folate. The onset of anemia is usually insidious and gradually progressive.

2. Glossitis :

Typically, the patient has a smooth, beefy, red tongue.

3. Neurologic manifestations :

Vit. B₁₂ deficiency particularly in patients of pernicious anemia, is associated with significant neurological manifestations in the form of subacute combined, degeneration of the spinal cord and peripheral neuropathy. While folate deficiency may occasionally develop neuropathy only. The underlying pathologic process consists of demyelination of the peripheral nerves, the spinal cord and the cerebrum signs and symptoms include numbness, paraesthesia, weakness, ataxia, poor finger coordination and diminished reflexes.

4. Others :

Mild jaundice, angular stomatitis, purpura, melanin pigmentation, symptoms of malabsorption, weight loss and anorexia.

(4) APLASTIC Anemia :

Aplastic anemia is defined as pancytopenia (ie. simultaneous presence of anemia, leucopenia and thrombocytopenia) resulting from aplasia of the bone marrow. The red bone marrow is reduced and replaced by fatty tissues. The underlying defect in all cases appears to be sufficient reduction in the number of hematopoietic pluripotent stem cells which

makes them unable to divide and differentiate. But, the RBCs are normocytic and nonmochronic.

Etiology and Classification :

Based on the etiology, aplastic anemia is classified into 2 main types: primary and secondary.

A. Primary aplastic anemia :

1. Fanconis anemia (congenital).

Autosomal recessive inheritance.

2. Immunologically mediated (acquired).

B. Secondary aplastic anemia :

1. Drugs :

(i) Dose – related aplasia eg. with antimetabolites (methotrexate), mitotic inhibitors (daunorubicin), alkylating agents (busulfan), nitrose urea, anthracyclines.

(ii) Idiosyncratic aplasia eg: with chloramphenol sulfa drugs, oxyphenbutazone, phenylbutazone, chlorpromazine, gold salts.

2. Toxic Chemicals eg : Benzene derivatives, insecticides, arsenicals

3. Infection eg : infectious hepatitis, EB virus infection, AIDS, other viral illness

4. Miscellaneous eg : associated with SLE and therapeutic X-rays

Clinical features :

The onset may occur at any age and is usually insidious. The clinical manifestations include :

1. Anemia and its symptoms like mild progressive weakness and fatigue.
2. Hemorrhage from various sites due to thrombocytopenia such as from the skin, nose, gums, vagina, bowel and occasionally in the CNS and retina
3. Infections in the mouth and throat are commonly present.
4. The lymph nodes, liver and spleen are generally not enlarged.

ANEMIA OF CHRONIC DISEASES :

Anemia of chronic diseases is the second common type of anemia. It is characterized by short life span of RBC's caused by disturbance in iron metabolism or resistance to erythropoietin action. Anemia develops after few months of sustained disease.

Anemia in chronic disorders is usually normocytic normochromic but can have mild degree of microcytosis and hypochromia unrelated to iron deficiency.

1. Anemia in chronic infections / inflammations

a) Infections eg : tuberculosis, lung abscess, pneumonia, osteomyelitis, subacute bacterial endocarditis, pyelonephritis.

b) Non – infections inflammations eg : Rheumatoid arthritis, SLE, vasculitis, dermatomyositis, scleroderma, sarcoidosis, Crohn's disease.

c) Disseminated Malignancies eg : Hodgkin's disease, disseminated carcinomas and sarcomas.

2. Anemia of renal disease :

Eg : Uraemia, renal failure

3. Anemia of hypometabolic state :

Eg : Endocrinopathies (myxoedema, Addison's disease, hyperthyroidism, hypopituitarism) protein malnutrition, scurvy, pregnancy, liver disease.

The severity of anemia is usually directly related to the primary disease process. The anemia is corrected only if the primary disease is alleviated

Investigations :

Hemoglobin estimation CBC

Peripheral blood film examination.

Red cell indices

(MCV, MCH & MCHC)

Leucocyte and platelet count

Reticulocyte count

ESR

Biochemical investigations

Blood Sugar

Blood Urea

Serum cholesterol

Serum Protein

Serum iron

Total iron binding capacity (TIBC)

MATERIALS AND METHODS

MATERIAL :

A clinical trial on vatha pandu was carried out in Govt. Siddha Medical College, Palayamkottai.

The 40 cases with clinical signs and symptoms of Vatha Pandu of both sexes of difference age (17-75) groups were selected and studied cases were selected in the department of maruthuvam (OP and IP) clinical parameters.

Cases were selected from out-patient and in-patient ward and the parameters of my care selection were

Symptoms

Looking pale, fatigue, swelling of the body and oedema, feeling of weakness, dry skin, white sclera, general malaise, numbness, glossitis, dyspnoea on exertion. Palpitations, heart murmur, angular stomatitis with soreness, angina, intermittent claudication of the legs, head ache, female abnormal menstruation, decreased energy.

Clinical Examination

Patients were subjected to physical examination on siddha methodology piniyari muraimai. It has 3 main principles which are Poriyalarithal pulanalarithal, vinathal and effected through “Envagai thervugal”.

Coming to the modern methodology a detailed clinical history was recorded and also physical examination was carried out.

All the cases were subjected to following investigation.

- Nadi paritchai, Neerkuri, Neikuri.

- Complete blood analysis TC, DC, Hb.

Total RBC, peripheral blood smear, E.S.R, PCV, MCV, MCH, MCHC.

- Routine urine analysis albumin,sugar,deposit.
- Stools for ova, cyst and occult blood.

Bio chemical analysis of serum cholesterol, blood urea and blood sugar was carried out.

Method of Treatment

Siddha system of medicine is based on mukkutra theory and hence the treatment is mainly aimed to bring down the three doshas to its equilibrium, there by restoring the physiological condition of the three dhoshas.

விரேசனத்தால் வாதந் தாமும்

வமனத்தால் பித்தந் தாமும்

நசிய அஞ்ஞனத்தால் ஐயந்தாமும்

It is necessary to treat the worm infestation, as is the prime Aetiology for Anaemia “M.V. pills was given (Dose -1- tablet early morning with hot water for deworming the patient.

Trial Medicine :

Treatment should be given to neturalise the vitiated mukkutram. The trial medicine used in the present study is

1. Thiratcha Choornam – 2gm இருவேளை பசுநெய்யுடன்

This medicine was given orally after meals. This medicines was prepared in the postgraduate gunapadam pratical hall with the knowledge

and guidelines of staffs by P.G. Maruthuvam department of Govt. Siddha Medical College, Palayamkottai.

Biochemical analysis and pharamacological studies of the drug was done to know the active principles and actions of trial medicine.

1. Sex Distribution

Table – 1 : Illustrates the Sex distribution and its relative percentage

Sl. No	Sex	Out patients		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Male	6	30	1	5
2.	Female	14	70	19	95

2. Age Distribution

Table – 2 : Illustrates the Age distribution and its relative percentage

Sl. No	Age group in Years	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	21-30	-	-	4	20
2.	31-40	3	15	4	20
3.	41-50	3	15	8	40
4.	51-60	6	30	4	20
5.	61-70	7	35	-	-
6.	Above 70	1	5	-	-

Above table Shows, the incidence is high in age between 61 – 70.

3. Kaalam

Table – 3 : Illustrates the Kaalam and its relative percentage

Sl. No	Kaalam	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Vatha Kaalam	4	20	-	-
2.	Pitha Kaalam	14	70	15	75
3.	Kapha Kaalam	2	10	5	25

From the above study, the maximum number of cases were treated in Pitha Kaalam.

4. Constitution of the body

Table – 4 : Illustrates the Constitution of the body and its relative percentage

Sl. No	Constitution of the body	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Vatha Thegi	-	-	-	-
2.	Pitha Thegi	-	-	-	-
3.	Kapha Thegi	-	-	-	-
4.	Thontha Thegi	20	100%	20	100%

In all, the patients had Thontha Thegi.

5. Gunam

Table – 5 : Illustrates the Gunam and its relative percentage.

Sl. No	Gunam	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Sathuva Gunam	-	-	-	-
2.	Rajo Gunam	20	100%	20	100%
3.	Thamo Gunam	-	-	-	-

In this present study cent percent cases belongs to Rajogunam.

6. Religion

Table – 6 : Illustrates the Religion and its relative percentage

Sl. No	Religion	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Hindu	16	80	17	85
2.	Christian	3	15	2	10
3.	Muslim	1	5	1	5

In this study, the maximum number of patients were Hindus.

7. Type of work

Table – 7 : Illustrates the Type of work and its relative percentage

Sl. No	Type of work	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Sedentary worker	-	-	-	-
2.	Moderate worker	17	85	20	100
3.	Hard worker	3	15	-	-

8. Socio Economic Status

Table – 8 : Illustrates the Socio Economic and its relative percentage

Sl. No	Socio Economic Status	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	High Class	17	85	15	75
2.	Middle Class	3	15	5	25
3.	Low Class	-	-	-	-

From the above study, economically poor class peoples were prone to Anaemia.

9. Habits

Table – 9 : Illustrates the Habits and its relative percentage

Sl. No	Habits	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Alcoholic	1	5	0	-
2.	Smoker	3	15	1	5
3.	Tabacco Chewer	0	0	0	-
4.	None	16	80	19	95

10. Dietary Pattern

Table – 10 : Illustrates the Dietary Pattern and its relative percentage

Sl. No	Dietary Pattern	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Mixed Diet	15	75	16	80
2.	Vegetarian	5	25	4	20

From the above study most of the patients were taken mixed diet.

11. Paruva Kaalam

Table – 11 : Illustrates the Paruva Kaalam and its relative percentage

Sl. No	Paruva Kaalam	Months	Out patientes		In patients	
			No. of Cases	Percentage	No. of Cases	Percentage
1.	Kaarkalam	Aavani, Purattasi	4	20	6	30
2.	Koothir Kaalam	Iyppasi, Karthigai	7	35	-	20
3.	Munpani Kaalam	Markazhi, Thai	-	-	-	-
4.	Pinpani Kaalam	Masi, Panguni	-	-	-	-
5.	Elavenil Kaalam	Chitirai, Vaikasi	-	-	-	-
6.	Muthuvenil Kaalam	Aani, Aadi	9	45	14	70

The above table shows most of the patients were treated in Muthuvenil and Karkalam.

12. Thinai

Table – 12 : Illustrates the Thinai and its relative percentage

Sl. No	Thinai	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Kurinji	-	-	-	-
2.	Mullai	-	-	-	-
3.	Marutham	20	100%	20	100%
4.	Neithal	-	-	-	-
5.	Paalai	-	-	-	-

Above the table shows most of the patients were from marutha nilam.

13. Weight Distribution

Table – 13 : Illustrates the Weight and its relative percentage

Sl. No	Weight Distribution	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Over Weight	1	5	-	-
2.	Ideal Weight	16	80	18	90
3.	Low Weight	3	15	2	10

14. Clinical Manifestations features :

Table – 14 : Illustrates the clinical manifestations and its relative percentage

Sl. No	Clinical Manifestations	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Palloriness of skin mucous membrane	20	100	20	100
2.	Fatigue / weakness	20	100	20	100
3.	Dyspnoea on Exertion	20	100	20	100
4.	Palpitation	4	20	5	25
5.	Constipation	10	50	12	60
6.	Head ache	9	45	8	40
7.	Angular Stomatitis	3	15	7	35

15. Kosam :

Table – 15 : Illustrates the Kosam and its relative percentage

Sl. No	Kosam	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Annamaya Kosam	20	100	20	100
2.	Pranamaya Kosam	20	100	20	100
3.	Manomaya Kosam	-	-		
4.	Vingnanamaya Kosam	-	-		
5.	Ananthamaya Kosam	-	-		

In all the patients, Annamayakosam, Pranamaya Kosam affected.

16. Disturbances in Vatha:

Table – 16 : Illustrates the Disturbances in Vatha and its relative percentage

Sl. No	Vatham	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Pranan	12	60	14	70
2.	Abanan	8	40	7	35
3.	Viyanan	6	30	5	25
4.	Udhanan				
5.	Samanan	6	30	7	35
6.	Nagan	-	-	-	-
7.	Koorman	-	-	-	-
8.	Kirukaran	10	50	12	60
9.	Devathathan	-	-	-	-
10.	Dhananjeyan	-	-	-	-

Above table shows most of the cases had altered Abanan, pranan, viyanan, samanan & kirukaran.

17. Disturbances in Pitham:

Table – 17 : Illustrates the Disturbances in Pitham and its relative percentage

Sl. No	Vatham	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Analapitham	-	-	-	-
2.	Ranjagapitham	20	100	20	100
3.	Prasakapitham	20	100	20	100
4.	Alosagapitham	-	-	-	-
5.	Sathagapitham	-	-	-	-

Above the table shows most of the cases had altered Ranjagapitham, Prasakapitham.

18. Disturbances in Kapham:

Table – 18 : Illustrates the Disturbances in Kapham and its relative percentage

Sl. No	Vatham	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Avalambagam	12	60	14	70
2.	Kilethagam	-	-	-	-
3.	Pothagam	7	35	6	30
4.	Tharpagam	-	-	-	-
5.	Santhigam	7	35	6	30

Above the table shows most of the cases had altered avalambagam, santhigam, & Pothagam.

19. Involvement of Ezhu Udalkattugal:

Table – 19 : Illustrates the Involvement of ezhu Udalkattugal and its relative percentage

Sl. No	Vatham	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Saaram	20	100	20	100
2.	Seneer	20	100	20	100
3.	Oon				
4.	Kozhuppu	-	-	-	-
5.	Enbu	7	35	6	30
6.	Moolai	-	-	-	-
7.	Sukkilam / Suronitham	-	-	-	-

From the above study all the patients had altered Saaram,seneer.

20. Envagai Thervugal:

Table – 20 : Illustrates the Condition of Envagai Thervugal and its relative percentage

Sl. No	Vatham	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Naadi	20	100	20	100
2.	Sparisam	-	-	-	-
3.	Naa	-	-	-	-
4.	Niram	20	100	20	100
5.	Mozhi	-	-	-	-
6.	Vizhi	-	-	-	-
7.	Malam	10	50	12	60
8	Moothiram	-	-	-	-

All the patients showed Pitha Kalapu Naadi (Pitha Vatham and Vathapitham).

21. Neerkuri - Neikuri:

Table – 21 : Illustrates the Neerkuri - Neikuri and its relative percentage

Sl. No	Vatham	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Neerkuri (Straw yellow in Colour & Clear)	20	100	20	100
2.	Neikuri				
	a. Aravena neendathu-vatham	3	-	6	30
	b. Aazhi pol paraviyathu-pitham	17	85	14	70
	c. Muthothu nindrathu-kabam				
	d. Thonthaneer				

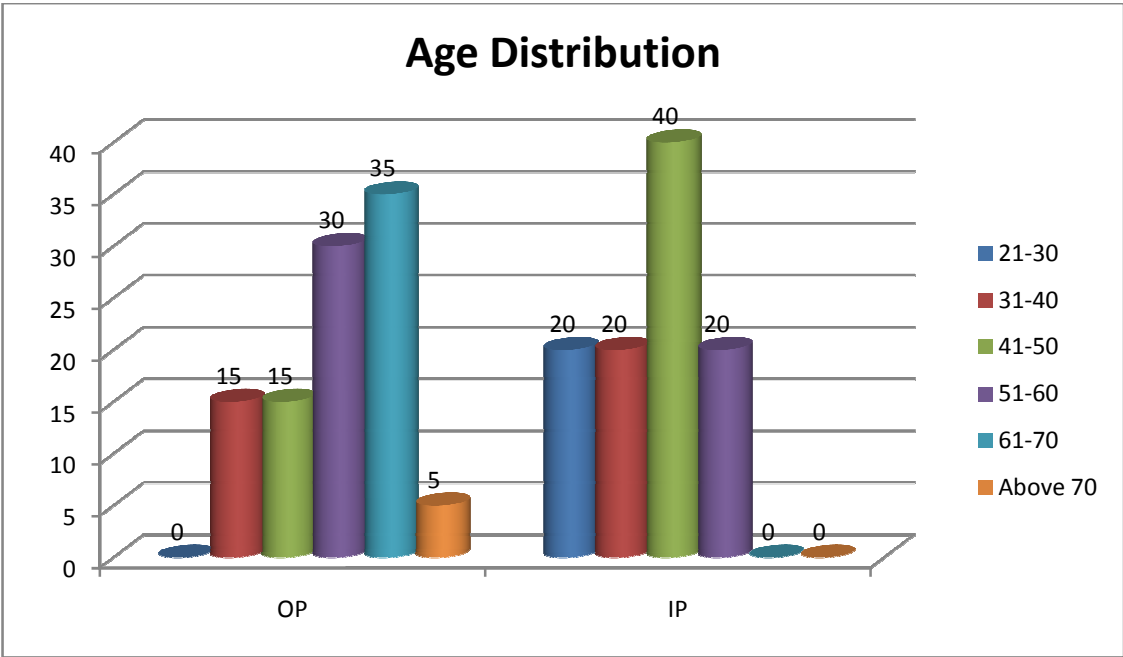
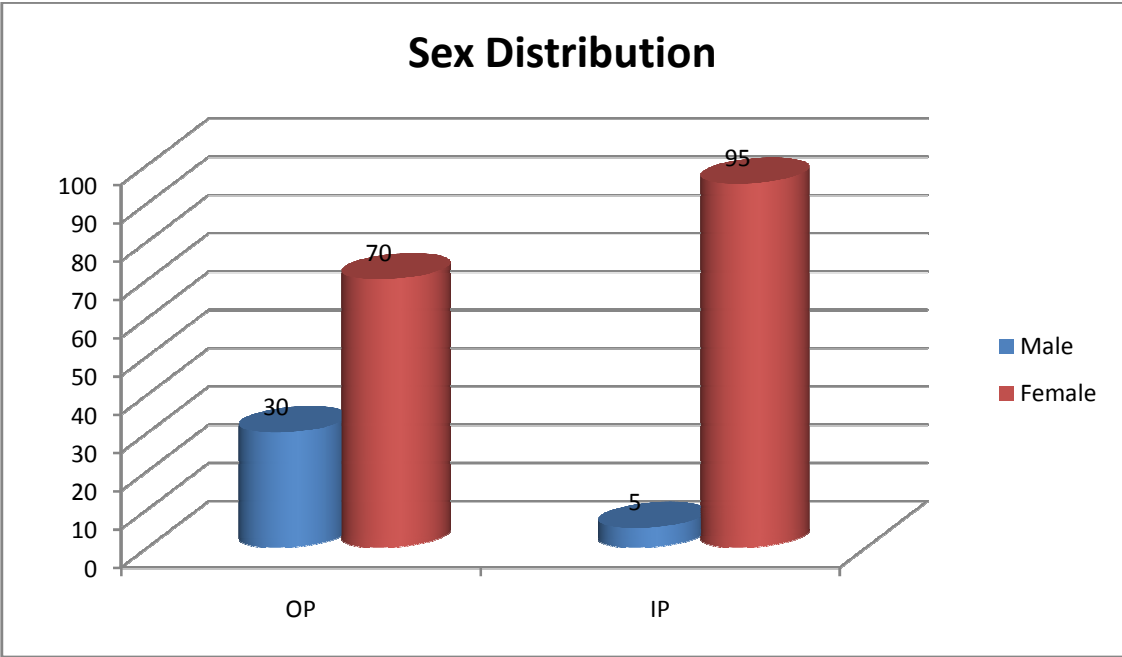
In this present study of neerkuri showed straw yellow in colour and . Most the patients had Patha Neer.

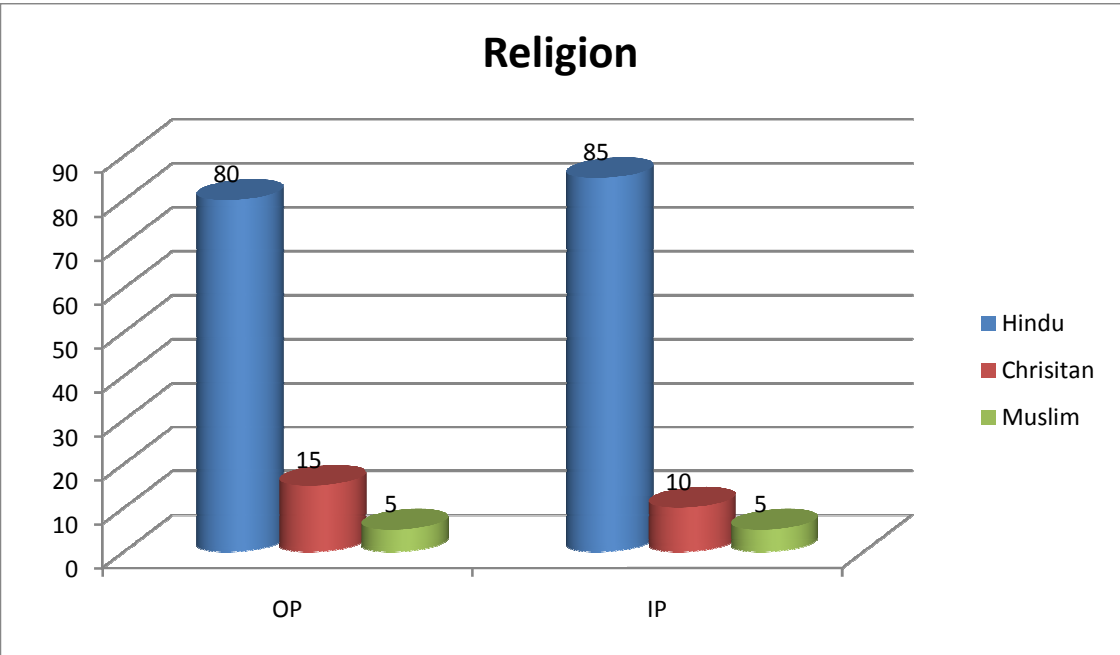
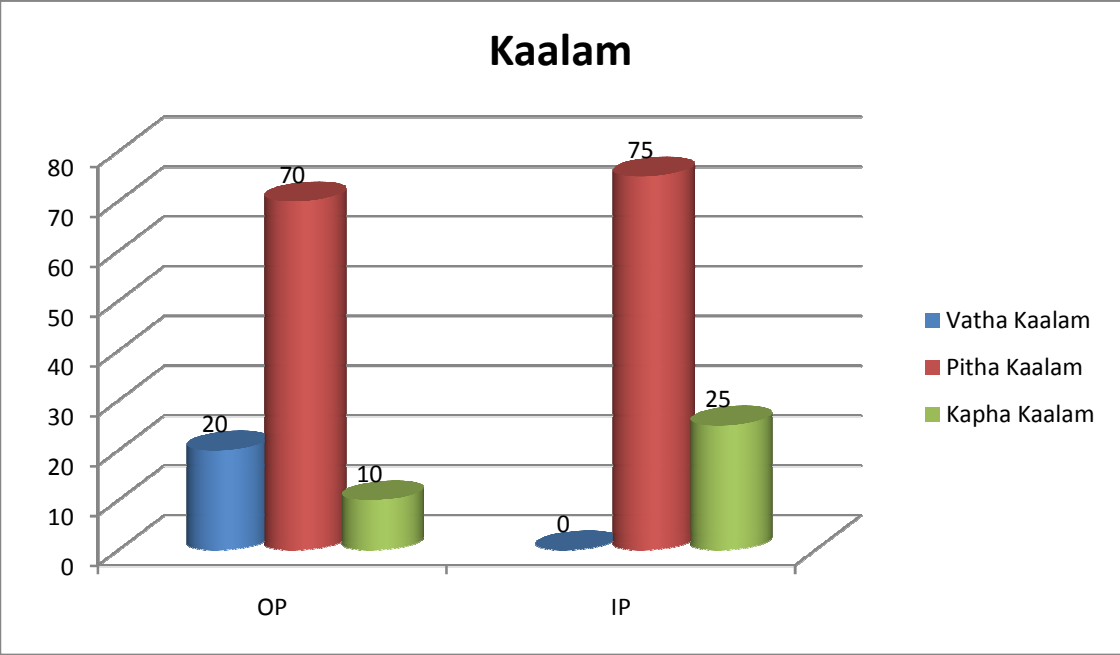
22. Assessment of Result:

Table – 22 : Illustrates the Assessment of Results and its relative percentage

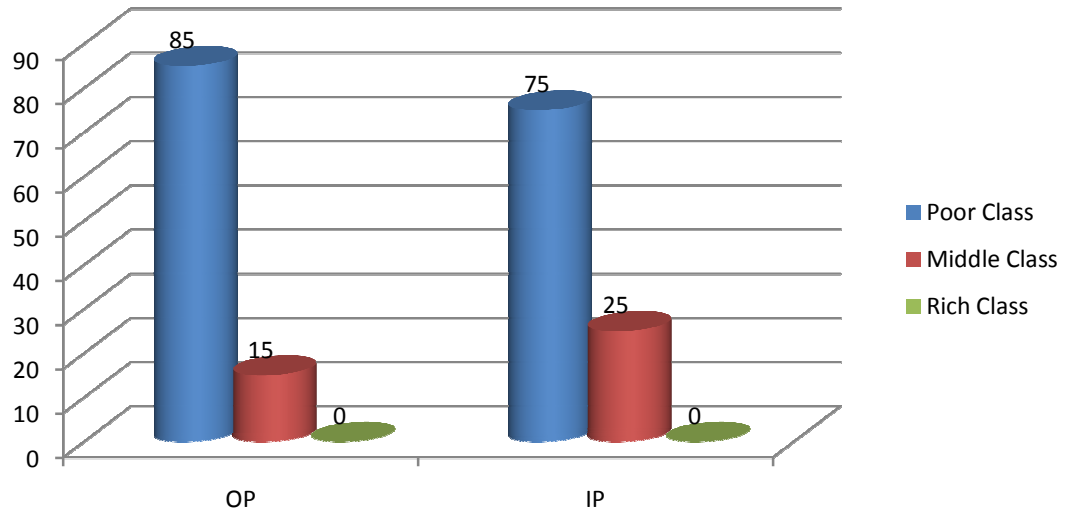
Sl. No	Grade	Out patientes		In patients	
		No. of Cases	Percentage	No. of Cases	Percentage
1.	Good	10	50	14	70
2.	Fair	7	35	4	20
3.	Poor	3	15	2	10

Most of the patients good results. At the end of the treatment the inpatients were discharged and they were advised to follow up treatment in P.G. Department of Maruthuvam O.P. for further management.

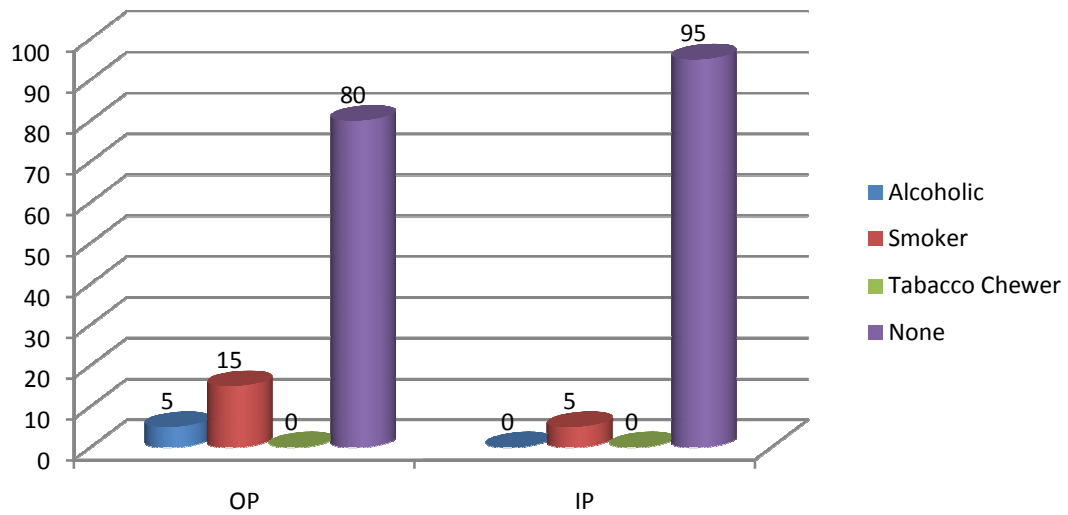


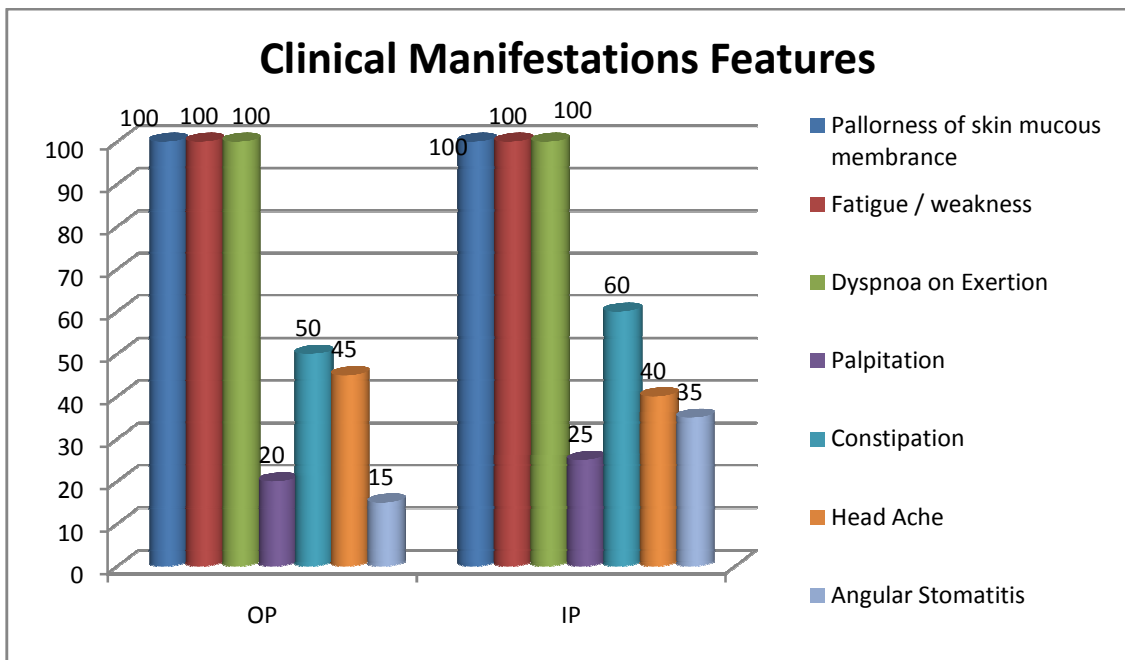
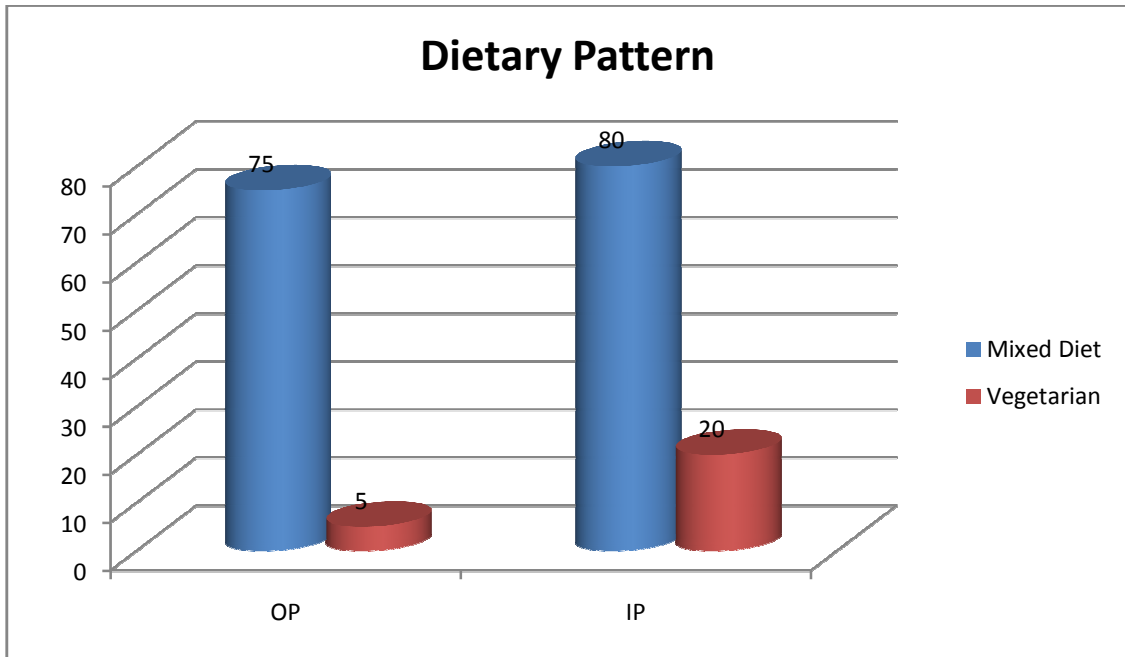


Socio Economic Status

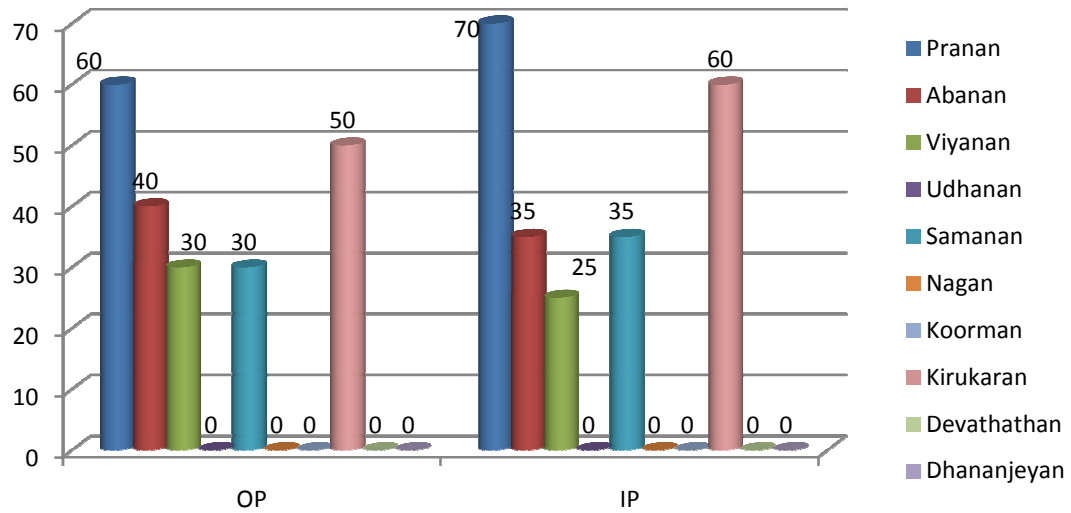


Habits

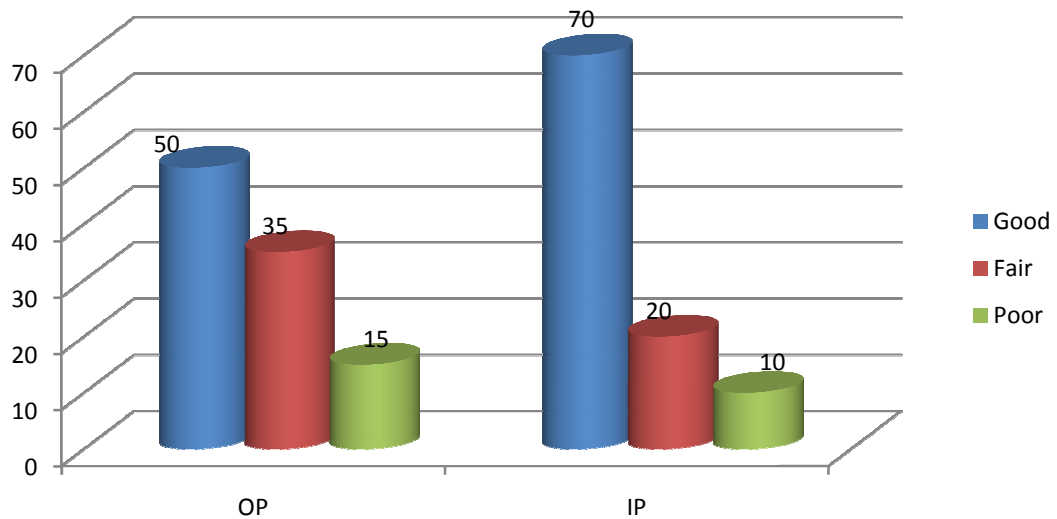




Disturbances in Vatha



Assessment of Result



RESULTS

Anaemia is corrected by the administration of medicinal iron. The response is evident within 10 days as improved well being and return of appetite, reduction of various symptoms.

Results were observed on the basis of two main criteria one of the basis of clinical improvement and other results derived from the blood picture. For all practical purpose the beneficial response of Thiratcha Choornam (internally) correcting anaemia may be taken as a suggestive evidence of anemia retrospectively. Results were derived on the basis of the following investigate parameter.

1. Haemoglobin – in gram / dl
2. Packed cell volume (percentage)
3. Hb% - in gram / dl
4. Mean corpuscular Volume – MCV (fl)
5. Mean corpuscular haemoglobin – MCH (piccogram)
6. Mean Corpuscular Haemoglobin Concentration (gm/dl) clinical improvement.

1. Breathlessness on exertion was present in all cases was not present in all cases after treatment like wise.
2. Feeling of weakness was present in all cases, before treatment and not present after treatment.
3. Palloriness of skin, mucous membrane was present in all cases, It was Moderately reduced in 16 cases after treatment.
4. Palpitation was present in nine patients moderately reduced in all cases after treatment.
5. Constipation was present in 22 patients was not present in all cases after treatment.
6. Head ache was present in 17 patients and was not present in 17 patient after treatment.
7. Angluar stomatitis was present in 10 patients and was not present in 10 patients after treatment.

Hematological Report :

1. At the time of admission haemoglobin percentage was decreased.
RBC count was low in majority of the cases.
2. In minority of patient MCV is also reduced.

3. No of days treated was 48 days, the efficacy was established through this period.
4. The raise of haemoglobin was good. (ic varied in range between 1gm – 3gm)

DISCUSSION

A small group of 40 patients with complaints of vatha pandu were selected. The diagnosis was again established and confirmed with the help of Envagai Thervugal, modern biochemical investigations like total red blood corpuscles count, haemoglobin percentage, MCHC, MCH, MCV, PCV, peripheral blood smear. These cases were given the trial drug.

During the course of treatment, the patient was subjected to routine investigation in urine, Albumin, sugar, deposits, sp. Gravity in blood urea. serum cholesterol, serum protein, in stools ova, cyst, in order to exclude other disorders. Eventhough anaemia is present in other systemic disorders. infectious disease (e.g. T.B) and with other connective tissue disorders they were excluded.

Age / Sex

The patients selected for my study were between 17-75 years, of both sexes. It depends upon various etiological factors. Regarding sex in this study, occurrence ratio is comparably more in Female than Male.

Occupation, Study and Habit

As shown in table No.2, 32 patients were from poor socio economic status and 8 from middle class family. Diet with rich nutrient fruits, milk, egg is closely binds to earning of an individual's income.

Etiology

It is observed that nutritional deficiency was noted in all the 40 cases. A well- balanced diet should include a sufficient amount of vegetables fruits for organic minerals and vitamins, together with other elements in a balanced proportion. Proper care of food and the manner and environment in which it is produced is of great importance in the preservation of health. In this nine patients who are strict vegetarians and due to their poor socio-economic status and ignorance of nutritious diet, they tends to take poor diet, which leads to vatahpandu noi. This is shown in table No 4. PANDU NOI occurs irrespective of religion.

Thinai & Kalam

According to table No. 11 the present study is contrary to what has been mentioned by many ancient siddhars. Marutham is a non-prevalent zone of any diseases, but various environmental changes and changes in the lifestyle in marutham, disease vathapandu noi, which occurs irrespective of

any season. Most of the cases subjected to this study were done in Muthu Vehil Karkalam. Aggravation of signs and symptoms were not observed in any season.

SUMMARY AND CONCLUSION

The research on “Vatha Pandu” was chosen with an intention to give solace to the patients who are suffering from this disease without any noxious effects.

Various literatures having relevant reference to the disease, vatha pandu were collected from both Siddha system as well as in modern system of medicine.

The efficacy of the trial drug “Thiratcha chooranam” was studied and observed during research.

Pandu an ancient clinical entity, with its historical importance correlates to the modern clinical entity Anaemia in every aspects. In “Vatha Pandu” noi chest discomfort, fatigue, dyspnoea on exertion, Palpitation headache palloriness of the skin, tongue, mucous membrane of the lips, palms and hands and conjunctiva, tachycardia are noted. The clinical features observed by various Siddhars perfectly match with clinical features discussed in western text books without any doubt.

The feasibility of considering vatha pandu as anaemia is observed after considering the siddha etiological factors which coincide to the modern aspects.

The diagnosis is established by siddha and modern methodology revealed that Nutritional Deficiency is common cause for America. The incidence is slightly greater in females than males due to their excessive physiological needs in menstrual cycle, lactation, pregnancy.

Regarding age incidence, this disease occurs at any age.

It'll be seen that sound diet is essential for overcoming disease. Proper care of food and the manner and environment in which it is produced is of great importance in the preservation health.

The clinical trial conducted with 40 patients.

The trial drug is easily tolerable by patients without any untoward side effects.

Biochemistry and pharmacological analysis reveal that the test drugs have significant haematinic effect.

CONCLUSION

The dissertation medicine Thiratacha Choornam and is a potent haematinic drugs for the remedy of Vatha pandu (Anaemia).

Research findings shows that 75% of patients were completely cured and also proved clinically.

The trial drug was very effective to the patients and there was no recurrence of symptoms. Cost of the drug is very cheap and free from side effects. So they are useful for long term purpose.

If a man strictly adhere to the immutable laws derived by Siddhars. He weill lead a healthy and happy life.

ANNEXURE I

PHARMACOLOGICAL ANALYSIS OF TRIAL MEDICINES

THIRATCHA CHOORNAM

சேரும் சரக்குகளும் அளவும் :

1	முந்திரிப்பருப்பு (ANACARDIUM OCCIDENTALE)	- 1 Balam (35 mgs)
2	பேரிச்சங்காய் (PHONEX DACTILIFERA)	- 1 Balam (35 mgs)
3	அதிமதுரம் (GLYCYRRHIZA GLABRA)	- 1 Balam (35 mgs)
4	கோரைகிழங்கு (CYPERUS ROTUNDUS)	- 1 Balam (35 mgs)
5	கொத்தமல்லி (CORIANDRUM SATIVAM)	- 1 Balam (35 mgs)
6	அல்லிக் கிழங்கு (NYMPHAEA NOUCHALI)	- 1 Balam (35 mgs)
7	சாதிக்காய் (MRISTICA FRAGRANS)	- 1 Balam (35 mgs)
8	இலவங்கம் (SYZYGIU MAROMATICUM)	- 1 Balam (35 mgs)
9	சுக்கு (ZINGIBER OFFICIANALE)	- 1 Balam (35 mgs)
10	விளாமுச்சு (PLECTRANTHUS VETTIVERUIDES)	- 1 Balam (35 mgs)
11	சிறுநாகப்பூ (MESUA NAGASSARIUM)	- 1 Balam (35 mgs)
12	இலவங்கப்பட்டை(CINNAMOMUM VERUM)	- 1 Balam (35 mgs)
13	சந்தனம் (SANTALUM ALBUM)	- 1 Balam (35 mgs)
14	மூங்கிலுப்பு (BAMBUSA ARUNDINACEAE)	- 1 Balam (35 mgs)
15	தாளிசம் (ABIES SPECTABILIS)	- 1 Balam (35 mgs)
16	நன்னாரி (HEMIDESMUS INDICUS)	- 1 Balam (35 mgs)
17	தக்கோலம் (ILLICIAM VERUM)	- 1 Balam (35 mgs)

- 18 நெல்லிவிதை (SEEDS OF PHYLLANTHUS EMBLICA) - 1 Balam (35 mgs)
- 19 நெற்பொரி (FRIED ORYZA SATIVA) - 1 Balam (35 mgs)
- 20 இலவங்கப்பத்திரி (CHINNAMOMUM TAMALA) - 1 Balam (35 mgs)
- 21 திப்பிலி (PIPER LONGUM) - 1 Balam (35 mgs)
- 22 சர்க்கரை - 1 Balam (35 mgs)

திராட்சை சூரணம் சேரும் சரக்குகள் :

சரக்குகளின் சுத்தி முறை :

1. முந்திரிப் பருப்பு - இளவறுப்பாக வறுத்து எடுத்துக் கொள்ள வேண்டும்.
2. பேரிச்சங்காய் - கொட்டை நீக்கி எடுத்துக் கொள்ள வேண்டும்.
3. அதிமதுரம் - தண்ணீரில் கழுவி மேல்தோல் நீக்கி சிறு துண்டு வெயிலில் உலர்த்த வேண்டும்
4. கோரைக்கிழங்கு - ஈரப்பதம் நீங்கும் அளவு வெயிலில் உலர்த்தி எடுக்க வேண்டும்.
5. அல்லிக் கிழங்கு - ஈரப்பதம் நீங்க வெயிலில் உலர்த்தி எடுக்க வேண்டும்.
6. வினா முச்சு - ஈரப்பதம் நீங்க வெயிலில் உலர்த்தி எடுக்க வேண்டும்.
7. சிறுநாகப்பூ - இளவறுப்பாக வறுத்த எடுக்க வேண்டும்.
8. இலவங்கப்பட்டை - ஈரப்பதம் இல்லாதவாறு வெயிலில் உலர்த்தி எடுக்க வேண்டும்.

9. சந்தனம் - ஈரப்பதம் இல்லாதவாறு வெயிலில் உலர்த்தி எடுக்க வேண்டும்.
10. தாளிசம் - ஈரப்பதம் இல்லாதவாறு வெயிலில் உலர்த்தி எடுக்க வேண்டும்.
11. நன்னாரி - மண்போக கழுவி வெயிலில் காயவைத்து எடுக்க வேண்டும்.
12. தக்கோலம் - ஈரப்பதம் இல்லாதவாறு வெயிலில் காயவைத்து எடுக்க வேண்டும்.
13. நெல்லிவிதை - ஈரப்பதம் இல்லாதவாறு வெயிலில் காயவைத்து எடுக்க வேண்டும்.
14. நெற்பொறி - உமி நீக்கி எடுத்துக் கொள்ள வேண்டும்.
15. இலவங்கப்பத்திரி - ஈரப்பதம் இல்லாதவாறு வெயிலில் உலர்த்தி எடுத்துக் கொள்ள வேண்டும்.
16. திப்பிலி - இளவறுப்பாக வறுத்து எடுத்துக் கொள்ள வேண்டும்.
17. கிராம்பு - மொட்டுப்போல் உள்ள பூவை எடுத்தெறியவும்.
18. சாதிக்காய் - மேல்தோல் நீக்கி நெருப்பில் வறுத்தெடுக்கும்.
19. சுக்கு - மேல்தோல் நீக்கி சுண்ணா நீரில் 1 மணி நேரம் ஊறவைத்து எடுக்கவும்.
20. தாளிசபத்திரி - இளவறுப்பாக வறுத்தெடுக்கவும்.
21. கொத்தமல்லி விதை - வறுத்து நன்றாய் புடைத்து எடுக்கவும்.
22. சர்க்கரை - குரணத்திற்கு சமஅளவு சர்க்கரை

செய்முறை :

சுத்தித்த எல்லா சரக்குகளையும் கல்உரலில் இட்டு வடித்து சலித்து, அனைத்தையும் ஒன்றுபட கலந்து வைத்துக் கொண்டு சூரணத்திற்கு சமஅளவு சர்க்கரையை சேர்த்து அரைத்து எடுத்து காற்றுப் புகாத குப்பிகளில் அடைத்து வைத்துக் கொள்ள வேண்டும்.

அளவு : 2 கிராம்

அனுபானம் : நெய்

மருந்து உட்கொள்ளும் காலம் : காலை, மாலை 1 மண்டலம்.

தீரும் நோய்கள் : பாண்டு, சோகை, இரத்தபித்தம் காமாலை, தாகசுரம், சயித்தியம்

- அகஸ்தியர் பரிபூரணம் 400

1. முந்திரிபருப்பு : B.N : Anacardium Occidentale
- குடும்பம் : Anacardiaceae
- பயன்படும் உறுப்பு : விதை
- சுவை : இனிப்பு, தன்மை – தட்பம், பிரிவு - இனிப்பு
- செய்கை : உரமாக்கி
- காமம்பெருக்கி

பித்த சுட்டையும், ஆண்மை பெருக்கையும், மந்தத்தையும் உண்டாக்கும்.

2. பேரிச்சங்காய் : B.N : Phoenix Dactylifera

குடும்பம் : பால்மே

பயன்படும் உறுப்பு : பழம்

சுவை : இனிப்பு, தன்மை – வெப்பம், பிரிவு –

கார்ப்பு

பொதுகுணம் :

நீரைச் சுரப்பிக்கின்ற பித்தம், மூர்ச்சை சுரம், நீர் கோவை, நீர்வேட்கை, இரத்தபித்தம், மதுமேகம், வாயிவைப்பு, அரோசகம், மலபந்தம் ஆகியவைகளைக் கண்டிக்கும்.

It contains Fe - 7.3mg / 100 gm and has found it to be a rich source of vitamin 'C' (3mg / 100gm)

- Wealth of India

3. தக்கோலம் : B.N : Illicium Verum

குடும்பம் : Magnoliaceae

சுவை : துவர்ப்பு, தன்மை – வெப்பம்

பிரிவு : கார்ப்பு

செய்கை :

வாதமடக்கி - Antivatha

துவர்ப்பி - Astrigent

வெப்பகற்றி - Febrifuge

உடலுரமாக்கி - Nutritive

பொதுகுணம் :

தக்கோலத்தில், பாண்டு, சுரம், வாயைப்பற்றிய வளி நோய்கள் போம், உடல் வன்மை பெறும், ஆண்மை உண்டாம். தளர்ந்த வெளியாகும் எருவைக் கட்டும்.

Alcohol Anethole is the cheif constituent.

4. அதிமதுரம் : Glycyrrhiza Glabra

குடும்பம் : Fabaceae

பயன்படும் உறுப்பு : வேர்

சுவை : இனிப்பு, கைப்பு

தன்மம் : வெப்பம்

பிரிவு : கார்ப்பு

செய்கை : வறட்சியகற்றி

உள்ளழலாற்றி

கோழையகற்றி

மலமிளக்கி

உரமாக்கி

பொதுகுணம் :

முப்பிணியால் வரும் புண், தாகம், நீர் வேட்கை, கண் நோய்கள், வெறி நோய், விக்கல், வெண்குட்டம், நீர் எரிச்சல், நஞ்சுகள், காமாலை, வெப்பு நோய்கள், ஐயத்தாலுண்டான கோளையை இழக்கச் செய்யும்.

5. கோரைக் கிழங்கு : *Cyperus rotundus*
குடும்பம் : *Cyperaceae*
செய்கை : துவர்ப்பி
வெப்பமுண்டாக்கி
உரமாக்கி
சிறுநீர் பெருக்கி
வியர்வை பெருக்கி
உள்ளழாற்றி

பொதுகுணம் :

இதனால் நளிரச்சுரம், குருதியழல் நோய், சுர வகைகள், நீர்வேட்கை, முப்பிணி, கழிச்சல், பத்திய தோடம், பித்தகாசம், கபரோகம், குதிக்காலைப் பற்றிய வாயு, வாந்தி இவை போகும்.

fat sugar gum, carbohydrate, essential oil, albuminous matter, starch
fibr and ash.

-Hand book on medicinal herbs

with uses.

6. கொத்தமல்லி : *B.N : Coriandru Sativum*
குடும்பம் : *Apiaceae*
சுவை : கார்ப்பு, தன்மை — சீதவெப்பம்,
பிரிவு : கார்ப்பு
செய்கை : பசித்தீத்தூண்டி

அகட்டுவாய் அகற்றி

வெப்ப முண்டாக்கி

சிறுநீர்ப் பெருக்கி

பொதுகுணம் :

உட்கூடு, நளிர்ச்சுரம், பயித்திய நோய், செரியாமை, வாந்தி, விக்கல், நாவறட்சி, பெருஏப்பம், புண் இவைகளை போம்.

The tried material contain ether extract - 3.12 ,Albuminoids – 24.46 ,Solubla Carbohydrate – 43.3 ,Woody fibre – 9.75 and Ash – 19.37,

7. சாதிக்காய் : B.N : Myristica Fragrans

குடும்பம் : Myristicaceae

சுவை : துவர்ப்பு, கார்ப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

பயன்படும் உறுப்பு : காய்

செய்கை : வெப்ப முண்டாக்கி

அகட்டுவாய் அகற்றி

மூர்ச்சை உண்டாக்கி

மணமூட்டி

காமம் பெருக்கி

உரமாக்கி

பொதுகுணம் :

இதனால் விந்து குறைவு, பெருங்கழிச்சல், வாயுவினால் உண்டாகும் நோய் தலைவலி, இரைப்பு, இருமல், நாட்பட்ட கழிச்சல், வெப்பத்தை முன்னிட்டு வரும் பிணிகள் இவைகள் போம்.

8. இலவங்கம் : Syzygium Aromaricum

குடும்பம் : Myrtaceae

சுவை : காரம், விறுவிறுப்புள்ளது

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

செய்கை : இசிவகற்றி

அகட்டுவாய்கற்றி

பசித்தீத்தூண்டி

பொதுகுணம் :

கிராம்பு, மயக்கம், பேதி, வாந்தி, குருதிக் கழிச்சல், நாட்பட்ட கழிச்சல், எருவாய் கடுப்பு, செவிநோய், சிவந்தமச்சம், கறுத்த மச்சம், கண்ணில் பூ, படைகள் தீரும்

moisture – 25.2, protein – 5.2, fat – 8.9, fibre-9.5, carbohydrate -46, and mineral matter – 5.2%, Calcium -740, Phosphorus -100, and iron – 4.9mg, iodine-50mg, vitamin – carotene thamine-0.08, and riboflavin-0.13mg, nicotinic acid is also present.

-The Hand Book on medicinal herbs with uses

9. அல்லிக் கிழங்கு : Nymphaea Nouchali, Brum
குடும்பம் : Nymphaeaceae
சுவை : சிறுதுவர்ப்பு
தன்மை : தட்பம்
பிரிவு : இனிப்பு
பொதுகுணம் :

கிழங்கை உட்கொள்ள, மேக அனல், உட்டினம், தாகம் ஆகிய வெப்ப ரோகங்கள் தணியும்.

10. நன்னாரி : Hemidesmus Indicus. Linn
குடும்பம் : Asclepiadaceae
பயன்படும் உறுப்பு : வேர்
சுவை : இனிப்பு, சிறு கைப்பு
தன்மை : தட்பம்
பிரிவு : இனிப்பு
பொதுகுணம் :

அழல் நோய், வண்டுகடி போம், மேலும் நீரேற்றம், நீர்வேட்கை, வாய்நீர் சுரப்பி, புணர்ச்சியின் குடு, நீரிழிவு, கிரந்தி, சுரவேட்கை இவை தணியும்.

Air roots – Essential Oil P-Methoxy Salicylic aldehyde as the major constituents. Others B-Sitosterol, and amyirin, lupuol terracyclic trtpene alcohols, small amount of resin, fatty acids, tannins, scaponin, a glycoside

11. இலவங்கப் பத்திரி : Cinnamomum Famala

குடும்பம் : Lauraceae

சுவை : கார்ப்பு, வீரியம் - வெப்பம்

பிரிவு : கார்ப்பு

செய்கை : வெப்பமுண்டாக்கி

அகட்டுவாய் கற்றி

பசித்தீத்தாண்டி

வியர்வை பெருக்கி

பொதுகுணம் :

மேகசுரம், ஐயசுரம், வெட்டை, இரைப்பை, இருமல், நீர்வேட்கை
குழல், வாந்தி, வாய்ப்புண், மேகக்கட்டி, தாது நட்டம், கைப்புச் சுவை
இவைகளைப் போக்கும்.

Cinnamic, acid, linaloy eugenol, Acitrate, Benzaldehylyde, Camphor,
Cadinane, Terpeneol, Geraniol, Pebzol acetate

12. தாளிசப்பத்திரி : Ables Spectabilis

குடும்பம் : Pinaceae

பயன்படும் உறுப்பு : இலை

சுவை : கார்ப்பு, தன்மை, வெப்பம்,

பிரிவு : கார்ப்பு

செய்கை : பசித்தாண்டி

அகட்டுவாய்கற்றி

கோழையகற்றி

உரமாக்கி

பொதுகுணம் :

கழிச்சல், சுரம் நாட்பட்ட இருமல், இரைப்பு, வாந்தி, வாய்வு, அசீரணம், அத்திசுரம் இவைகளுக்கு வழங்கலாம். இதனால் சுகப்பிரசவமுண்டாகும்.

The Leaves contain nitrogen 1.44, and Ash – 4.06, the also contain and alkaloid 0.16% and essential oil.

-The Hand book on medicinal herbs with uses.

13. திப்பிலி : Piper longum

குடும்பம் : Piperaceae

பயன்படும் உறுப்பு : காய், அரிசி

சுவை : உலர்ந்தது, தன்மை – வெப்பம்

பிரிவு : இனிப்பு

செய்கை : வெப்பமுண்டாக்கி

அகட்டுவாய் அகற்றி

பொதுகுணம் :

ஈளை, இருமல், இரைப்பு, உப்பிசம் முதலிய பிணிகளை போக்கத் தக்கவாறு திப்பிலியை ஆளவேண்டியதாம்.

N hexadecane n- heptadecane n-octa decane n- nanodeana n-eicosane
terpinonane zingiperane symaney phenathlyalchol

14. மூங்கிலுப்பு : Bambusa arudinaceae

குடும்பம் : Bambusaceae

பயன் :

இது மூங்கில் கணுக்களில் தங்கி நிற்கும். இதில் 4 கிராம் எடைத் திப்பிலி 2 கிராம், ஏலம் 1 கிராம், இலவங்கம் ½ கிராம், சர்க்கரை 8 கிராம் பொடித்துக் கலந்து, வேளைக்கு 4 கிராம் எடை வீதம், ஐய நோய், இருமல், இரைப்பு முதலியவைகளுக்குக் கொடுக்கலாம்.

Protein fat carpuhodrat minerals, calcium phosphorus, iron, magnesium, sodium, copper, Chlorin, Vitamin, Riboflavin niacin, choline.

15. நெற்பொறி : Oryza Sativa Linn

பயன்படும் உறுப்பு : அரிசி, தவிடு, உமி

சுவை : இனிப்பு, தன்மை – தட்பம், பிரிவு - இனிப்பு

பொதுகுணம் :

நெல்லைப் பொரித்து வழி போக்கி எடுத்து, கஞ்சி காய்ச்சி நோயாளிகளுக்கும் கொடுக்கலாம். முக்கியமாய் வயிற்று நோய், வெள்ளை, நீர்சருக்கு, சுரம் முதலிய நோயாளருக்கு இது வன்மை தரும். நீர் வேட்கை, வனம், மந்தம், பெருவயிறு, கழிச்சல், சுவையின்மை முதலிய நோய்களும், மூர்ச்சையும் நீங்கும்.

16. சிறுநாகப்பூ : Mesua naga Ssarium

குடும்பம் : Clusiaceae

பயன்படும் உறுப்பு : மொக்கு பூ

சுவை : சிறுகைப்பு துவர்ப்பு

தன்மை : தட்பம்

பிரிவு : கார்ப்பு

செய்கை : துவர்ப்பி

அகட்டுவாய் அகற்றி

பொதுகுணம் :

நீரடைப்பு, குருதிப் போக்கு, புண் கொப்புளம், கால்எரிச்சல்

ஆகியவை போக்கும்.

17. சந்தனம் : Santalum Album

குடும்பம் : Santalaceae

பயன்படும் உறுப்பு : மரம், எண்ணெய்

சுவை : கைப்பு, சிறுதுவர்ப்பு,

தன்மை : தட்பம், வெப்பம்

பிரிவு : இனிப்பு, கார்ப்பு

செய்கை : உடற்றேற்றி

சிறுநீர்ப்பெருக்கி

வியர்வைப் பெருக்கி

வெப்பமுண்டாக்கி

அழுகல்மணமகற்றி

துவர்ப்பி

குளிர்ச்சியுண்டாக்கி

பொதுகுணம் :

இதனால் முக்குற்றம், மனக்கலக்கம், உட்குடு, ஒழுக்கு வெள்ளை, நாவறட்சி, நமைச்சல் இவை நீங்கும். உடல் வன்மை பெறும். மருந்துகளுக்குச் செந்நிறமூட்டவும் இந்திர கோபத்திற்குப் பதிலாகவும் செஞ்சந்தனத்தைக் கையாளுவதுண்டு.

18. சுக்கு : Zingiber Officinale

குடும்பம் : Zingiberaceae

பயன்படும் உறுப்பு : கிழங்கு (உலர்ந்தது)

சுவை : கார்ப்பு, தன்மை – வெப்பம்

பிரிவு : கார்ப்பு

செய்கை : வெப்பமுண்டாக்கி

பசித்தீத்தூண்டி

அகட்டுவாய்வகற்றி

பொதுகுணம் :

சுக்கினால் செரியாமை, மார்பெரிச்சல், புளியேப்பம், வெப்பம், கீழ்வாய் நோய், இரைப்பு, இருமல், கழிச்சல், நீரேற்றம், குன்மம், வயிற்றுப்பிசம், காதுக் குத்தல், முகநோய், தலை நோய், குலைவலி, பாண்டு, வயிற்றுக் குத்தல், ஐயசுரம் போம்.

19. இலவங்கப்பட்டை : Cinnamomum Verum Presi
குடும்பம் : Myrtaceae
சுவை : காரமும் இனிப்புமுடையது
தன்மை : தட்பம்
பொதுகுணம் :

இது பாம்புக்கடி, சிலந்திப்பூச்சிக்கடி முதலியவைகளின் நஞ்சைப் போக்கும். இஃது இரைப்பு, இருமல், வயிற்றுக்கடுப்பு, உள்மூலப் புண் இவைகளைப் போக்கும். உடற்குக் குளிர்ச்சியை உண்டுபண்ணும்.

20. நெல்லி விதை : Phyllanthus Emblica
குடும்பம் : Euphorbiaceae
பயன்படும் உறுப்பு : விதை
சுவை : புளிப்பு, துவர்ப்பு, இனிப்பு
செய்கை : குளிர்ச்சி உண்டாக்கி
சிறுநீர்ப் பெருக்கி
மலமிளக்கி
குணம் :

வெறி நோய், ஐய நோய், பீனிசம், வாய்நீர்ச் சுரப்பு, வாந்தி, மயக்கம், தலைசுற்றல், மலபந்தம், பிரமேகம் இவை போகும்.

21. விளாமுச்சு : Plectranthus vettiveroides

குடும்பம் : Labiaceae

பயன்படும் உறுப்பு : வேர்

சுவை : கசப்பு, தன்மை – சீதம், பிரிவு - இனிப்பு

செய்கை : குளிர்ச்சி உண்டாக்கி

பித்தமடக்கி

குணம் :

விளாமுச்சு வேருக்கு நீரிழிவு, கண்ணீரிழிவு, குருதி அழல்நோய்,

நீர்வேட்கை, வெறி மயக்கம், மூர்ச்சை சோபை தலைவலி தீச்சுரம் போம்.

22. சர்க்கரை

ANNEXURE II

BIO-CHEMICAL ANALYSIS OF THRATCHA CHOORANAM

PREPARATION OF THE EXTRACT

5 gms of the drug was weighed accurately and placed in a 250ml clean beaker. Then 50ml of distilled water is added and dissolved well. Then it is boiled well for about 10 minutes. It is cooled and filtered in a 100ml volumetric flask and then it is make upto 100ml with distilled water. This fluid is taken for analysis.

QUALITATIVE ANALYSIS

S.No	Experiment	Observation	Inference
1	TEST FOR CALCIUM 2ml of the above prepared extract is add 2ml of 4% ammonium oxalate taken in a clean test tube. To this Solution.	A white precipitate is formed	Indicates the presence of Calcium.
2	TEST FOR SULPHATE 2ml of the extract is added to 5% Barium chloride solution.	A white precipitate is formed	Indicates the presence of sulphate
3	TEST FOR CHLORIDE A white precipitate is formed The extract is treated with Silver nitrate Solution.	A white precipitate is formed	Indicates the presence of chloride

4	TEST FOR CARBONATE The substance is treated with Concentrated HCL	No Brisk effervescence is formed	Absence of Carbonate
5	TEST FOR STARCH The extract is added with weak iodine solution.	Blue Colour is formed	Indicates the presence of starch
6	TEST FOR FERRIC IRON The extract is acidified with Glacial acetic acid and add Potassium ferro cyanide	No Blue colour is formed	Absence of Ferric Iron
7	TEST FOR FERROUS IRON The extract is treated with Concentrated nitric acid and Ammonium thic cyanide solution.	Blood red colour is formed	Indicates the presence of ferrous Iron
8	TEST FOR PHOSPHATE The extract is treated with ammonium Molybdate and concentrated nitric acid	No yellow precipitate is formed	Absence of Phosphate
9	TEST FOR ALBUMIN The extract is treated with Esbatch's Reagent	No yellow precipitate is formed	Absence of Albumin
10	TEST FOR TANNIC ACID The extract is treated with ferric Chloride	No blue black precipitate formed	Absence of Tannic acid
11	TEST FOR UNSATURATION Pottassium permanganate	It gets decolourised	Indicates the presence of an

	solution is added to the extract		saturated compound
12	<p>TEST FOR THE REDUCING SUGAR</p> <p>5ml of Benedict's qualitative Solution is taken in a test tube and allowed to boil for 2 mins and add 8-10 drops of the extract and again boil it for 2 mins</p>	colour change occurs	Indicates the presence of Reducing sugar
13	<p>TEST FOR AMINO ACID</p> <p>One or two drops of the extract is placed on a filter paper and dried it well. After drying 1% Ninhydrin is sprayed over the same and dried it well.</p>	Violet Colour is formed	Indicates the presence of Amino Acid
14	<p>TEST FOR ZINC</p> <p>The extract is treated with Pottassium ferrocyanide</p>	No white precipitate is formed	Absence of Zinc
15	<p>TEST FOR THE COPPER :</p> <p>The extract is treated with acetic acid and potassium Ferro cyanide are added.</p>	A brown precipitate is formed	Copper is conformed.

ANNEXURE III

PHARMACOLOGICAL ANALYSIS OF TRIAL MEDICINES

Study on the Haematinic effect of

Thiratcha Choornam

Variety of preparations in siddha system of medicine are well known for its haematinic effects, of which Thiratcha Choornam is one of the best. To provide the efficacy of Thiratcha Choornam an attempt was made to study its effect using “Albino rats”. For this purpose, rats were made anaemic by the following procedure.

Artificially Induced Anaemia

The albino rats taken for this experiment, were kept in aluminum cages and provided with drinking water and milk. The administration of the Thiratcha Choornam under investigation was started, when the hemoglobin level fell to 6 to 6.5 gram / 100ml. At the beginning of the experiment. RBC count, Hb% were determined.

I. Study of Rates

The albino rats were first divided in two equal groups, with three rats in each group. The first group received water. The 2nd group received the drug at a dose of 100 mg / 100gm body weight. All the above procedures

were continued for six weeks in once a day dose. The Haemoglobin levels or rats were measured I, II, III, IV weeks. The results observed are tabulated in the following chart.

STUDY OF HAEMATINIC ACTION

Thiratchai Chooranam

Serial No	Drugs	Dose	Before drug Administration				After Drug Administration			Remarks
			Initial Reading	Ist Week	IIInd Week	III Week	IV week	V week		
1	Control (Water) 2ml	2 ml	5.8	5.8	5.6	5.5	5.3	5.2	5.4	
			6.2	6.2	6.0	6.0	5.7	5.5		
			6.0	6.0	5.7	5.6	5.3	5.1		
			6.4	6.4	6.1	6.0	5.7	5.5		
			6.2	6.2	6.0	5.9	5.6	5.5		
			6.5	6.5	6.2	6.1	5.8	5.6		
2	Tiratchai Choorana mam	100 mg	6	6.0	6.2	6.5	7.1	8.5	8.6	
			6.2	6.4	6.7	6.7	7.4	8.6		
			6.5	6.2	6.5	6.7	7.4	8.4		
			6.4	6.4	6.6	6.8	7.5	8.7		
			6.2	6.4	6.7	6.7	7.8	8.9		
			6.6	6.8	6.9	7.0	7.5	8.7		

Result :

The trial drug Thiratcha Choornam has got significant haematinic action.

ANNEXURE IV
POST GRADUATE RESEARCH CENTRE
GOVT. SIDDHA MEDICAL COLLEGE PALAYAMKOTTAI
MARUTHUVAM POTHU – BRANCH – I
PROFORMA OF VATHA PANDU
(ANAEMIA)

Name of the medical unit :	Nationality :
I.P No :	Religion :
Name :	Date of Admission :
Age :	Date of Discharge :
Sex :	No. of days treated:
Occupation :	Diagnosis :
Income :	Results :
Address :	Medical Officer :

Complaints & Duration :

H/O Present Illness :

H/O previous Illness :

Personal History :

Family History :

CLINICAL EXAMINATION - SIDDHA SYSTEM

PORIPULANGAL :

Sensation :

T ate :

Sight :

Smell :

Touch :

KANMEDRIYAM :

Kai :

Kaal :

Vaai :

Eru Vaai :

Karu Vaai :

Gunam :

Sathuvam :

Rajo :

Thamo :

Nilam :

Kurinchi :

Mullai :

Marutham :

Neithal :

Palai :

YAKKAI :

Vatham :

Pitham :

Kabam :

Kalappu :

PARUVAKALAM:

Kar :

Koothir :

Munpani :

Pinpani :

Elavenil :

Mudhuvenil :

UTKAYAM, ADAHAKAYAM :

Puyam :

Sayam :

Kaal :

Patham :

Karuvaai :

PIRA URUPPUKALIN NILLAI :

Eruthyam :

Puppusam :

Kalleeral :

Manneeral :

Kudal :

Siruneeragam :

Mummalam :

Viyarvai :

Malam :

Moothiram :

MUKKUTRANGAL :

VATHAM :

Pranan :

Abanan :

Viyanan :

Uthanan :

Samanan :

Nagan :

Koorman :

Kirukaran :

Devathathan :

Dhananjeyan :

PITHAM :

Analam :

Ranjagam :

Pirasagam :

Aalosagam :

Sathagam :

KABAM :

Avalambagam :

Kilaethagam :

Pothagam :

Tharpagam :

Sandhigam :

ENVAGAI THERVUGAL :

Na :

Niram :

Mozhi :

Vizhi :
Sparisam :
Malam :
Niram :
Edai :
Ilagal :
MOOTHIRAM :
NEERKURI :
Niram :
Edai :
Manam :
Nurai :
Enjal :
NEIKURI :
Vatham :
Pitham :
Kabam :
NAADI :
EZHU UDARKATTUGL :
Saram :

Seneer :

Oon :

Kozhuppu :

Enbu :

Moolai :

Sronitham :

Sukkilam :

GENERAL EXAMINATION

Consciousness : General appearance :

Stature : Height :

Weight : Nourishment :

Skin Changes : Facies :

Anaemia : Jaundice :

Cyanosis : JVP :

Pedal Oedema : Engorged Venis :

Abdominal distension : Premature Cataract :

Congenital anomaly :

Temperature :

Pulse :	Rate	Rhythum	Volume	Character
---------	------	---------	--------	-----------

Blood pressure :	mm/Hg	(R)	(L)
------------------	-------	-----	-----

U.L.

L.L.

Respiratory Rate :

Heart Rate :

SYSTEMIC EXAMINATION

Examination of Cardiovascular System :

Examination of Respiratory System :

Examination of Abdomen :

Examination of Central Nervous System :

Laboratory Investigations

Blood

TC

DC P L E B M

Hb%

RBC Count

Platelet Count

Reticulocyte count

PCV

MCV

MCH

MCHC

Serum Protein

Serum Cholesterol

Blood Urea

Peripheral blood smear

ESR ½ hr

1 hr

Motion

Ova

Cyst

Occult Blood

URINE

Albumin

Sugar

Deposit

Bile Salt

Bile Pigment

SPECIAL INVESTIGATION

1. Bariummeal and endoscopy
2. Bone marrow examination
3. Skiagram
4. Sputum for AFB :
5. Radiological Investigation :

6. Ophthalmoscopic Examination :

7. E.C.G.

DIFFERENTIAL DIAGNOSIS :

CASE SUMMARY :

DIAGNOSIS :

PROGRESS NOTE :

Date	Complaints	Hb%	Wt.Kgs	Drug	Complications

**POST GRADUATE RESEARCH CENTRE
GOVT. SIDDHA MEDICAL COLLEGE PALAYAMKOTTAI
MARUTHUVAM POTHU – BRANCH – I
PROFORMA OF VATHA PANDU
(ANAEMIA)**

Name of the medical unit :

I.P No :

Name :

Date of Admission :

Age / Sex :

Date of Discharge :

Sex :

No. of days treated:

Occupation :

Diagnosis :

Income :

Results :

Nationality / Religion :

Medical Officer :

Patient condition on admission	Patient condition at the time of discharge
C/D Signs : G/E : Weight : Blood Pressure : Blood : Hb% TRBC % PCV MCHC MCV Serum Cholesterol : Blood Smear Serum Protein Urine : Alb : Sug : Dep : Bile pigment MOTION Ova Cyst Occult Blood :	C/D Signs : G/E : Weight : Blood Pressure : Blood : Hb% TRBC % PCV MCHC MCV Serum Cholesterol : Blood Smear Serum Protein Urine : Alb : Sug : Dep : Bile pigment MOTION Ova Cyst Occult Blood :

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- BAILEY AND LOVE’S SHORT PRACTICE OF SURGERY

S.No	I.P.No	Name	Age / Sex	No of days	Hb gm/d	RBC Million	PCV	MCV	MCH	MCHC	Peripher al blood	Hb gm/dl	RBC Million /	PCV	MCV	MCH	MCHC	Peripher al blood
1	2320	Ganapathi	70/M	48	5.7	2.5	17	68	22	33.5%	MH	7.9	2.51	21	83.6	27.8	33.3	N
2	2433	Thavamaniyammal	60/M	48	9.7	3.49	29	83	27.7	33.4%	N	11.4	6.6	32.2	88	31.6	35.6	N
3	2439	Thanus Kodi	63/M	48	9	4.88	27	61.6	20.5	33.3%	MH	10.5	4.3	30	69.76	24.4	35	N
4	2387	Shanumugam	43/M	48	8.8	3.76	27.8	73.9	23.4	31.7%	N	9.2	3.7	27	72.97	24.8	34	N
5	2453	Parvathi	52/F	48	10	4.3	30	91.4	23.7	26.0%	N	11.4	4.3	33	76	26.9	35	N
6	2471	Mari Packiyam	54/F	48	9.4	3.6	28	83	25.7	30.9%	N	11.4	3.68	34	92	30.9	33.5	N
7	2472	Chithirai Vadivoo	54/F	48	9.8	4.7	30	84	20	24.7%	N	11	4.7	33	70.2	23	33.3	N
8	2509	Santhi	42/F	48	9.8	4.28	29	87.6	22.9	26.1%	N	11.1	4.2	33	78.5	26.4	33.6	N
9	2521	Muruga vadivoo	50/F	48	9.5	3.61	33	92.8	26.3	28.4%	N	10.8	3.6	31	86	30	34.8	N
10	3037	Barathi	57/F	48	9.5	4.03	287	69	23.5	33.9%	MH	11	4.12	33	80	26	33.3	N
11	3052	Indhu Rani	53/F	48	9	4.2	27	62	21	33.3%	MH	10.2	4.29	30.4	71	20.9	29.3	N
12	3107	Pitchammal	75/F	48	8	3.5	24	68.5	22.5	33.3%	MH	9.5	3.5	28	74	26	31.9	N
13	3301	Babu Rav	66/M	48	9.8	3	28	70	32	34.0%		11.5	3	28	70	32	34	N
14	3761	Sabeena	33/F	48	9.8	3.5	29	83	27	23.2%	N	9.8	3.5	29	83	83	27	N
15	3762	Poomari	32/F	48	9.8	4.1	707	23.9	33	79.0%	N	11	4.17	33	79	79	26.3	N
16	3764	Vadivammal	70/F	48	11.1	3.5	33	74	28.5	33.9	N	11	3.5	33	94	31.4	33.3	N
17	3763	Durai	20/M	48	10	4.09	30	73.3	24.4	33.3%	MH	11.5	11.5	32	77	27.9	35	N
18	3814	Parvathi	65/F	48	8.5	4	25	62.5	21.5	34	MH	10	4	30	75	25	33.3	N
19	3815	Uchimagali	40/M	48	10	4	30	75	25	33.3%	N	11	4.01	33	82	27.4	33.3	N
20	3851	Mariammal	65/F	48	9.8	4	28	70	24	35.0%	N	11	4.12	33	82.5	27.5	33.37	N

S.No	I.P.No	Name	Age / Sex	No of days	Hb gm/d	RBC Million	PCV	MCV	MCH	MCHC	Peripher al blood	Hb gm/dl	RBC Million /	PCV	MCV	MCH	MCHC	Peripher al blood
1	50927	Muppidathi	44/F	48	9.6	4.8	31.2	65	20	30.8%	MH	11.5	4.8	33	70.8	23.5	34	N
2	50928	Panjali	48/F	48	7.2	3.79	25.4	67	19	28.3%	MH	9.1	3.79	27	71	24	33	N
3	50686	Mariyammal	26/F	48	9.8	3.41	30	88.3	28.7	32.6%	N	11.4	3.4	33	97	33	34.5	N
4	52382	Chandra	55/F	48	10	4	30	75	25	33.3%	MH	11	4	33	82.5	27.5	36.6	N
5	52399	Saroja	40/F	48	9.8	3.5	30	84	28	32.6%	N	11.7	4	35	87	29	33.4	N
6	55750	Shanmugathai	35/F	48	10	3	30	75	33.3	34.3%	N	11.5	3.2	36	103	35.9	34.8	N
7	55800	Suleka	40/F	48	9.5	3.6	28.5	79	26.38	33.3%	N	11.3	3.7	33	89	26.38	33.3	N
8	54089	Avudai Sundari	45/F	48	8	3.5	24	70	24.8	33.3%	N	9.7	3.5	28	80	27	34.6	N
9	59484	Murugeswari	26/F	48	6	2.5	7	72	33	33.3%	MH	9	2.7	27	100	33.3	33.3	N
10	59667	Valliammal	30	48	10	4	30	75	25	33.3%	N	12	4.2	36	85.7	28.5	33.3	N
11	59668	Sankarammal	56	48	9.2	3	27	90	30.6	34.0%	N	11	3.1	33	106	35	33.3	N
12	59695	Amirthakani	57/F	48	9	3	27	90	30	33.3%	N	11	3.1	33	100	35	33	N
13	59680	Anthoniammal	44/F	48	8.5	3	25.5	85	28.3	33.3%	N	11	2	27	100	33.3	33.34	N
14	73103	Jeyalakshmi	58/F	48	10	3.3	30	90	30.3	33.3%	N	12	3.36	36	167	35.7	33.3	N
15	73102	Sudha	23/F	48	10	3.3	30	90	30.3	33.3%	N	11.5	3.56	36	95.5	32.3	33	N
16	73879	Alagunayagi	42/F	48	8.4	3.6	24	81	22.8	33.3	N	10	3.6	34	95.5	32.3	33	N
17	84033	Kanirose	45/F	48	9	3	27	90	30	33.3%	N	11	3.25	33	96	33.8	33.3	N
18	86433	Gurubagavathi	40/F	48	8	4	24	80	26.6	32.2	N	10.2	4.1	30	73.1	24.8	31	N
19	86432	Ganesan	37/F	48	9	3	27	67.5	22.5	33.3%	N	10.2	3.1	32	103	34.5	33.4	N
20	59734	Muppidathi	30/F	48	7.8	4	24	60	19.5	32.5%	N	10	4.1	30	73.1	24.39	33.3	N

BEFORE TREATMENT

AFTER TREATMENT

Sl. No	IP No	Name	Age/Sex	TC cells Cum m	DC%			ESR		TC cells Cum m	DC%			ESR		Urine Analysis					
					P	L	E	1/2 hr	1 hr		P	L	E	1/2 hr	1 hr	Before Treatment			After Treatment		
1	2320	Ganapathi	70/M	5500	62	33	2	15	30	5400	62	31	1	5	10	N	N	N	N	N	N
2	2433	Thavamaniyammal	60/M	7000	48	46	4	12	24	6600	45	51	2	7	15	N	N	N	N	N	N
3	2439	Thanus Kodi	63/M	5000	62	28	7	12	24	5000	62	27	2	6	12	N	N	N	N	N	N
4	2387	Shanumugam	43/M	2200	60	33	4	7	14	2200	33	4	2	7	14	N	N	N	N	N	N
5	2453	Parvathi	52/F	5800	60	34	4	12	24	5800	60	34	2	6	12	N	N	N	N	N	N
6	2471	Mari Packiyam	54/F	7800	54	41	3	16	32	6900	53	40	1	10	20	N	N	N	N	N	N
7	2472	Chithirai Vadivoo	54/F	9500	56	36	5	10	20	9600	56	34	1	5	10	N	N	N	N	N	N
8	2509	Santhi	42/F	3600	48	41	8	10	20	3600	48	40	3	7	14	N	N	N	N	N	N
9	2521	Muruga vadivoo	50/F	9200	66	28	3	10	20	9200	66	27	2	5	10	N	N	N	N	N	N
10	3037	Barathi	57/F	6000	66	30	1	10	20	6000	66	30	11	5	10	N	N	N	N	N	N
11	3052	Indhu Rani	53/F	8800	60	30	0	10	20	8900	60	30	1	5	10	N	N	N	N	N	N
12	3107	Pitchammal	75/F	8600	57	37	6	10	20	8700	57	35	1	7	14	N	N	N	N	N	N
13	3301	Babu Rav	66/M	7600	65	28	1	10	20	7600	65	28	1	5	10	N	N	N	N	N	N
14	3761	Sabeena	33/F	5000	60	35	1	7	14	5000	60	34	1	7	14	N	N	N	N	N	N
15	3762	Poomari	32/F	8900	64	30	0	5	10	8900	63	29	0	5	10	N	N	N	N	N	N
16	3764	Vadivammal	70/F	7100	60	34	5	10	20	7100	60	32	2	5	10	N	N	N	N	N	N
17	3763	Durai	20/M	6400	62	33	1	5	10	6400	62	33	1	5	7	N	N	N	N	N	N
18	3814	Parvathi	65/F	6500	53	38	1	10	20	6500	53	38	1	5	10	N	N	N	N	N	N
19	3815	Uchimagali	40/M	6500	63	30	3	10	20	6000	60	31	10	7	14	N	N	N	N	N	N
20	3851	Mariammal	65/F	6500	62	53	3	10	15	6000	62	40	2	6	12	N	N	N	N	N	N

BEFORE TREATMENT

AFTER TREATMENT

Sl. No	IP No	Name	Age/Sex	TC cells Cum m	DC%				ESR		TC cells Cum m	DC%				ESR		Urine Analysis					
					P	L	E	M	1/2 hr	1 hr		P	L	E	M	1/2 hr	1 hr	Before Treatment			After Treatment		
1	50927	Muppidathi	44/F	9200	60	25	3	1	12	24	9300	70	35	2	1	10	20	N	N	N	N	N	N
2	50928	Panjali	48/F	6700	56	39	3	0	15	30	6700	55	39	2	0	7	14	N	N	N	N	N	N
3	50686	Mariyammal	26/F	9300	51	45	2	0	10	20	9200	50	45	1	0	5	10	N	N	N	N	N	N
4	52382	Chandra	55/F	7000	60	25	3	0	6	12	7000	59	24	1	0	6	12	N	N	N	N	N	N
5	52399	Saroja	40/F	5300	69	38	3	0	7	14	5300	69	37	1	0	6	12	N	N	N	N	N	N
6	55750	Shanmugathai	35/F	8700	65	32	3	0	11	22	8800	66	33	3	0	10	20	N	N	N	N	N	N
7	55800	Suleka	40/F	6500	50	46	4	1	13	26	6500	50	46	4	1	5	15	N	N	N	N	N	N
8	54089	Avudai Sundari	45/F	8500	68	27	5	0	8	20	8500	67	25	1	0	5	10	N	N	N	N	N	N
9	59484	Murugeswari	26/F	9000	66	32	22	0	6	15	9000	66	31	1	0	5	10	N	N	N	N	N	N
10	59667	Valliammal	30	9000	64	33	2	0	10	14	9100	67	33	2	0	7	14	N	N	N	N	N	N
11	59668	Sankarammal	56	6000	60	33	2	0	13	20	6100	61	33	2	0	5	10	N	N	N	N	N	N
12	59695	Amirthakani	57/F	9200	62	35	3	1	10	22	9200	62	35	1	1	17	14	N	N	N	N	N	N
13	59680	Anthoniammal	44/F	8500	64	33	2	0	11	24	8400	64	32	1	0	7	14	N	N	N	N	N	N
14	73103	Jeyalakshmi	58/F	8900	50	30	2	0	8	20	8900	50	30	3	0	5	10	N	N	N	N	N	N
15	73102	Sudha	23/F	8500	52	29	1	1	7	14	8500	49	28	1	1	8	16	N	N	N	N	N	N
16	73879	Alagunayagi	42/F	9600	50	30	1	1	9	18	9600	49	30	1	1	5	10	N	N	N	N	N	N
17	84033	Kanirose	45/F	8700	30	35	4	1	12	24	8700	50	33	4	1	5	10	N	N	N	N	N	N
18	86433	Gurubagavathi	40/F	7000	60	30	3	1	7	14	7000	60	30	1	1	5	10	N	N	N	N	N	N
19	86432	Ganesan	37/F	7600	60	28	1	0	5	10	7600	62	35	1	0	7	14	N	N	N	N	N	N
20	59734	Muppidathi	30/F	6500	62	53	3	1	10	45	6600	62	40	2	1	6	12	N	N	N	N	N	N

S.No	I.P.No	Name	AOD	DOD	NODT		TOTAL NODT	DRUG ADGVANT
					IP	FOLLOWE D OP		
1	2320	Ganapathi	18/7/12	18/9/12	48	0	48	2 gm T.C. with Ghee bd.
2	2433	Thavamaniyammal	26/7/12	19/9/12	48	0	48	3 gm T.C. with Ghee bd.
3	2439	Thanus Kodi	26/7/12	17/9/12	48	0	48	4 gm T.C. with Ghee bd.
4	2387	Shanumugam	26/7/12	17/9/12	48	0	48	5 gm T.C. with Ghee bd.
5	2453	Parvathi	27/7/12	19/9/12	48	0	48	6 gm T.C. with Ghee bd.
6	2471	Mari Packiyam	28/7/12	10/8/12	13	35	48	7 gm T.C. with Ghee bd.
7	2472	Chithirai Vadivoo	28/7/12	24/1/12	27	21	48	8 gm T.C. with Ghee bd.
8	2509	Santhi	30/7/12	13/9/12	43	5	48	9 gm T.C. with Ghee bd.
9	2521	Muruga vadivoo	1/8/12	25/8/12	26	22	48	10 gm T.C. with Ghee bd.
10	3037	Barathi	12/9/12	23/9/12	11	37	48	11 gm T.C. with Ghee bd.
11	3052	Indhu Rani	13/9/12	20/11/12	48	0	48	12 gm T.C. with Ghee bd.
12	3107	Pitchammal	19/9/12	17/10/12	28	20	48	13 gm T.C. with Ghee bd.
13	3301	Babu Rav	27/9/12	25/11/12	48	0	48	14 gm T.C. with Ghee bd.
14	3761	Sabeena	2/11/12	14/11/12	13	25	48	15 gm T.C. with Ghee bd.
15	3762	Poomari	2/11/12	11/11/12	10	38	48	16 gm T.C. with Ghee bd.
16	3764	Vadivammal	2/11/12	14/11/12	23	25	48	17 gm T.C. with Ghee bd.
17	3763	Durai	2/11/12	7/12/12	36	12	48	18 gm T.C. with Ghee bd.
18	3814	Parvathi	8/11/12	22/11/12	11	39	48	19 gm T.C. with Ghee bd.
19	3815	Uchimagali	8/11/12	3/12/12	26	22	48	20 gm T.C. with Ghee bd.
20	3851	Mariammal	15/11/12	13/12/12	29	19	48	21 gm T.C. with Ghee bd.

AOD - Date of Admission

DOD - Date of discharge

TC - Thiratchai Chooranam

NODO- Number of days Treated

S.No	I.P.No	Name	AOD	DOD	FOLLOWED OP	DRUG ADJUVANT
1	50927	Muppidathi	6/7/12	24/8/12	48	2gm T.C. with Ghee Bd
2	50928	Panjali	6/7/12	24/8/12	48	2gm T.C. with Ghee Bd
3	50686	Mariyammal	9/7/12	27/8/12	48	2gm T.C. with Ghee Bd
4	52382	Chandra	11/7/12	28/8/12	48	2gm T.C. with Ghee Bd
5	52399	Saroja	11/7/12	27/8/12	48	2gm T.C. with Ghee Bd
6	55750	Shanmugathai	23/7/12	10/9/12	48	2gm T.C. with Ghee Bd
7	55800	Suleka	23/7/12	10/9/12	48	2gm T.C. with Ghee Bd
8	54089	Avudai Sundari	3/8/12	21/10/12	48	2gm T.C. with Ghee Bd
9	59484	Murugeswari	4/8/12	22/9/12	48	2gm T.C. with Ghee Bd
10	59667	Valliammal	5/8/12	22/9/12	48	2gm T.C. with Ghee Bd
11	59668	Sankarammal	5/8/12	22/9/12	48	2gm T.C. with Ghee Bd
12	59695	Amirthakani	5/8/12	22/9/12	48	2gm T.C. with Ghee Bd
13	59680	Anthoniammal	6/8/12	24/9/12	48	2gm T.C. with Ghee Bd
14	73103	Jeyalakshmi	17/9/12	14/11/12	48	2gm T.C. with Ghee Bd
15	73102	Sudha	17/9/12	15/9/12	48	2gm T.C. with Ghee Bd
16	73879	Alagunayagi	20/9/12	8/11/12	48	2gm T.C. with Ghee Bd
17	84033	Kanirose	20/10/12	8/12/12	48	2gm T.C. with Ghee Bd
18	86433	Gurubagavathi	30/10/12	18/12/12	48	2gm T.C. with Ghee Bd
19	86432	Ganesan	30/10/12	18/12/12	48	2gm T.C. with Ghee Bd
20	59734	Muppidathi	6/8/12	23/8/12	48	2gm T.C. with Ghee Bd

AOD - Date of Admission

TC - Thiratchai Chooranam

DOD - Date of discharge